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SPECIFIC TISSUE REACTION TO PHOSPHOLIPIDS: A SUGGESTED EXPLANATION FOR THE SIMILARITY OF THE LESIONS OF SILICOSIS AND PULMONARY TUBERCULOSIS

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INTRODUCTION

MANY of the pathological lesions of silicosis and pulmonary tuberculosis are identical. Fortunately for the experimental pathologist, the lesions of experimental silicosis and tuberculosis, while not identical with, are similar to those of the actual diseases in most respects. Pathological lesions represent a characteristic tissue response to a definite type of stimulation. The similarity of the lesions in these conditions would indicate that they are produced by substances much more alike in nature than are the tubercle bacillus and quartz. An obvious inference is that inhaled or injected finely particulate quartz powder produces either a physical or chemical change in some substance normally present in the body, and that this altered substance stimulates the formation of the typical pathological lesions found in these conditions.

The microscopic appearance of the early lesions of experimental silicosis[1, 2] suggests that the monocyte is in some way responsible for the later fibrotic reaction. There is an extensive monocytic response to the inhalation or injection of finely powdered quartz with later destruction and disintegration of these cells. Normally, the lung alveolus rids itself of any finely particulate material by phagocytosis and transportation by monocytes. Particles under 5 microns in size, regardless of their nature, are engulfed by monocytes. The monocytes with their load of fine particles either move to the upper air passages and are coughed up or migrate into the lymph spaces, thence to the regional lymphatic nodes, where the particles are disposed of in different ways, depending upon the nature of the material. If the phagocytosed material is not toxic to the monocytes this process goes on indefinitely, as in anthracosis. If the phagocytosed material is toxic it will produce death and disintegration of the monocytes, resulting in an interruption of the normal dust clearance from the alveoli and the formation of certain characteristic pathological lesions.

When large amounts of fine quartz dust are either inhaled by or injected into the lungs of experimental animals there is the usual active monocytic response to particulate material, that is, the phagocytic cells invade the area and become engorged with dust particles. A number of them reach the upper air passages and are coughed up; others reach the lymph spaces and are transported to the lymphatic nodes; still others are held in the alveolar spaces. The silica, either by its physical or chemical action, changes the intracellular material, causing death of the cell with disintegration of the cell membrane and discharge of the intracellular material and the contained quartz particles. There is no evidence to show that the quartz is changed. It still has the ability to stimulate a further monocytic response. According to our hypothesis, the liberated intracellular material is the substance that initiates the typical fibroblastic reaction with formation of nodules.

The different diseases of lipoid metabolism, such as Gaucher's, Neumann-Pick's, Tay-Sach's, Hand-Schiller-Christian's, etc., all show varying degrees of fibrosis. Sabin[3] and her collaborators[4] have shown that of all the fractions of the tubercle bacillus the lipoids alone produce

tubercles. Of their various controls only one material tested acts just like tuberculo-phosphatide, namely, lecithin. These lipoids produce a marked new growth of general connective tissue as well as tubercular granulation tissue and tubercle-like structures. Monocytes are known to contain a considerable amount of lipoid material. The fact that the tissue response to the injection of quartz is mainly a fibroblastic reaction would suggest that the stimulating agent might be lipoidal in nature. For this reason the toxic substance was sought in the lipoid fraction of early silicotic lesions.

Three groups of experiments were performed to determine:

Experiment I.—The presence and nature of the lipoids in early silicotic lesions and the amounts in which they occur.

Experiment II.—The rate and amount of increase in the phospholipids in the lungs of rabbits which had been injected intratracheally with finely particulate silica.

Experiment III. — Whether or not the recovered phospholipid when freed of silica and re-injected into animals will produce tissue reactions comparable to the pathological lesions found in silicosis and tuberculosis.

In order to determine the nature of the lipoids in early silicotic lesions and the amounts in which they occur the following experiments were conducted.

EXPERIMENT I

Eight rabbits were each given 250 mg. of finely powdered quartz (the particles ranging in size from 0.5 to 3 microns) intratracheally, intraperitoneally and subcutaneously. Two rabbits were killed at intervals of 1, 2, 3 and 4 months. The lungs, peritoneal and subcutaneous nodules were removed and extracted separately with ether. The lungs of seven normal rabbits were each extracted in a similar manner as a control on the silica lungs.

Extraction of lipoids from the lungs.—The rabbits were anæsthetized with ether and bled from the carotids and jugulars. Each lung was removed at the hylus and as much blood as possible was expressed. They were weighed and this weight taken as "wet weight". Thin sections were taken for histological study. The lung tissue was ground as finely as possible with 40 g. of coarse sand. Five volumes of ether were added and the extraction repeated five times. The ether extract was filtered several times and evaporated. The lung residue was then extracted with absolute alcohol. The alcohol was evaporated. The lipoid obtained by alcohol extraction was redissolved in ether and added to the ether extract. The combined ethereal solutions were evaporated. The residue after evaporation will be referred to as "total lipoid". The extracted lung tissue and sand were dried and weighed. This weight plus the "total lipoid" minus the weight of the sand is called "dry weight" of lungs.

The "total lipoid" was redissolved in ether and filtered into ice-cold acetone. A heavy, white, flocculent precipitate formed. The precipitate was separated by centrifuging and pouring off the supernatant acetone. It was then evaporated until all the remaining acetone was removed. A wax-like, yellowish-brown, viscous, semi-solid material remained.

Results.—The recovered substance on analysis had the physical and chemical properties of a phospholipid. It was then soluble in ether, chloroform and benzene; insoluble in acetone; could be separated into two portions by precipitation in alcohol; formed a fine, even, permanent emulsion with water; could be precipitated from water by acetone; oxidized readily in air, turning brown and having a disagreeable odour. On hydrolysis it yielded fatty acids, phosphoric acid and nitrogen in the proportions found in the phospholipids. It appeared to be a mixture of lecithin and cephalin, or a closely related phospholipid. This material is referred to as "crude phospholipid".

Normal lungs of 9 to 12 g. "wet weight" and 2.5 to 3.8 g. "dry weight" yielded 200 to 290 mg. of lipoid from which an average of 50 mg. of crude phospholipid was obtained (see table). The lungs of rabbits 1 to 4 months after the intratracheal injection of finely powdered quartz yielded 480 to 1,100 mg. of lipoid containing 300 to 420 mg. of crude phospholipid. The peritoneal and subcutaneous nodules also contained considerable amounts of similar material. Pieces of subcutaneous tissue equal in weight to the nodules did not yield any acetone precipitate.

Conclusions.—The greatly increased amounts of phospholipids in the tissues of rabbits after injection of finely particulate quartz suggests that these substances were attracted to the regions by the presence of the quartz. Correlation of the histological picture and chemical findings suggests that the source of the phospholipid was the monocyte. The fact that this substance was found to be increasing with time and the number of monocytes containing quartz particles, seen in histological sections, naturally led to the following group of experiments.

The following experiments were performed to determine the rate and amount of increase in the phospholipids of rabbit lungs injected intratracheally with finely particulate silica. As a control, animals were injected in the same manner with equal amounts of kaolin, the particles of which were of corresponding size. It is an established fact that kaolin alone does not produce fibrosis.

EXPERIMENT II

Fourteen rabbits were injected intratracheally, each receiving 250 mg. of finely particulate quartz (0.5 to 3.0 microns) suspended in 2.5 c.c. of distilled water. Seven rabbits were given similar amounts of kaolin by the same method. Two of the silica-injected rabbits and one kaolin-rabbit were killed 1, 2, 3, 6, 12, 16 and 20 weeks after injection. The phospholipids were extracted from the lungs as described above.

Results.—The material injected, time interval, wet weight, dry weight, total lipoid and crude phospholipid are shown in the accompanying table. The rate and amount of increase in lung phospholipid are shown on the graph.

TABLE I.

Rabbit No.	*Material injected*	*Time interval*	*Wet weight*	*Dry weight*	*Total lipoid*	*Crude phospholipid*
Normal lungs	*mg.*		*g.*	*g.*	*mg.*	*mg.*
1	–	–	10.5	–	250	30
2	–	–	12.30	3.0	200	28
3	–	–	9.86	3.8	265	93
4	–	–	9.21	3.2	290	71
5	–	–	8.64	2.58	250	24
6	–	–	11.96	2.21	260	41
7	–	–	9.03	2.15	290	48
Pneumonic lungs						
8	–	–	41.34	4.58	480	64
9	–	–	19.67	3.86	280	134
SiO_2 lungs	SiO_2					
32	250 mg.	1 wk.	15.15		430	81
33	" "	1 "	11.50		390	95
34	" "	2 wks.	7.41	2.68	340	107
35	" "	2 "	7.60	2.13	320	55
37	" "	3 "	8.21	2.05	230	130
38	" "	3 "	15.08	5.65	240	200
39	" "	6 "	11.70	3.86	380	180
40	" "	6 "	9.30	3.50	480	380
41	" "	12 "	11.19	4.26	500	373
42	" "	12 "	10.48	3.34	580	352
43	" "	16 "	10.05	2.43	490	397
18 old	" "	16 "	14.56	5.36	720	420
44	" "	20 "	19.21	3.74	790	218
45	" "	20 "	21.18	4.25	580	227
Kaolin lungs	Kaolin					
46	250 mg.	1 wk.	24.80		350	30
47	" "	2 wks.	13.92	4.87	580	89
48	" "	3 "	15.78	6.90	730	160
49	" "	6 "	12.58	4.26	490	110
50	" "	12 "	9.93	3.72	290	121
51 (*pneumonic*)	" "	16 "	15.62	3.15	460	120

Conclusions.—There is a gradual increase in the amount of phospholipid present in the silica lungs for four months. This runs parallel with the histological picture, namely, phagocytosis of the silica, the breaking down of the monocytes, invasion of the area by fibroblasts and the early fibrous tissue reaction. After this there is a falling off in the amount of recoverable phospholipid.

The decreasing amount of phospholipid corresponds in time with the histological picture of fibrosis. The monocytes have disappeared from the central portions of the nodules. They have been replaced by typical fibroblasts. The newly-formed connective tissue is becoming hyalinized. The breaking down of monocytes has become limited to small numbers at the margins of the nodules, where there is still some active fibroblastic reaction. The original large amount of phospholipid has probably, after stimulating the growth of fibrous tissue, been removed or changed by this defense mechanism. The monocytes are now only attracted to the margins of the nodules, where small numbers are broken down and there is a slowly progressing stimulation of fibrous tissue growth. The change in the tissue reaction probably accounts for the decrease in the amount of phospholipid. The amount of phospholipid recovered from the lungs of rabbits appears to correspond with the number of disintegrating monocytes seen in the histological sections.

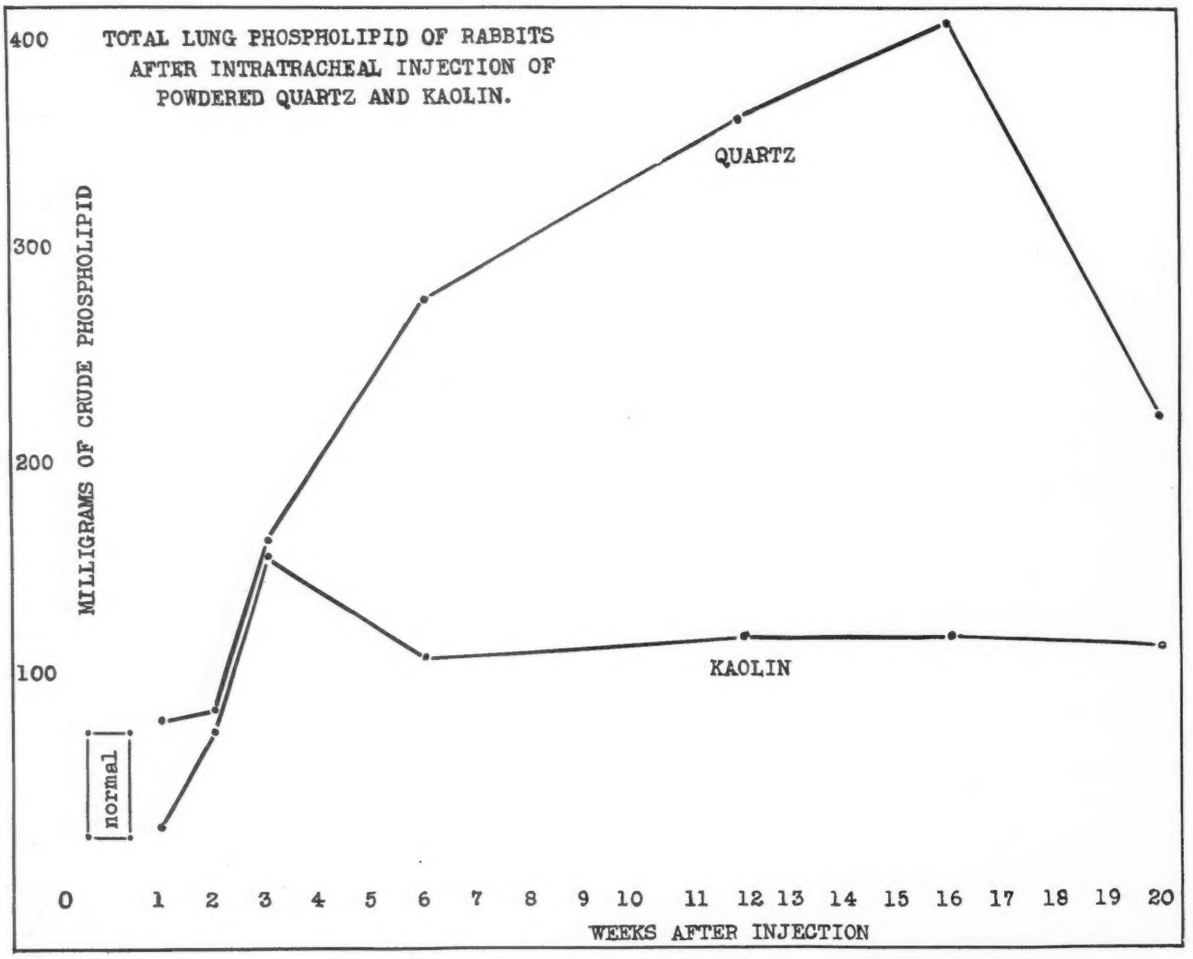
There is a small initial rise in the amount of phospholipid in the kaolin lungs. This is probably accounted for by the mere presence of the monocytes. The curve relating lung-phospholipid with time remains a straight line for months. The maintained slight elevation above the normal phospholipid level is probably accounted for by the engulfing of kaolin by monocytes and foreign-body giant-cells. These cells are seen in microscopic sections to remain in the lungs in increased numbers for long periods of time in a perfectly healthy state.

In the case of the animals receiving intratracheal injections of finely particulate quartz there is a marked increase in the phospholipid of all lungs showing the typical early tissue response. If our hypothesis (that the phospholipid liberated from the monocyte is responsible for the typical cellular reaction) is correct, injection of this phospholipid material obtained from the lungs should produce lesions comparable to those found in experimental silicosis and tuberculosis. The fact that Sabin and others have been able to produce fairly typical tubercles with the phospholipid fraction of tubercle bacilli and with pure lecithin would suggest that the greatly increased amounts of phospholipid present in the lungs after intratracheal injections of silica are responsible for the production of the fibrous nodules which so closely resemble tubercles.

The following experiments were performed to determine the tissue reaction to intraperitoneal injections of recovered, silica-free phospholipid.

EXPERIMENT III

The recovered phospholipid dissolved in ether was filtered to remove any particulate silica. Microincineration of the recovered phospholipid and acid treatment of the ash showed this material to be free from siliceous particles. Sabin's method of injection was followed in order that the lesions produced in our animals could be compared with those which she produced and so clearly described. Sabin, in her production of tubercles with the phospholipids of tubercle bacilli, gave daily intra-



peritoneal injections of 80 and 122 mg. for periods of from 1 to 15 days. The animals were then killed at intervals from twenty-four hours to six months after the last injection.

Experiment.—Daily doses of 18.5 mg. of recovered phospholipid in emulsion in 2.5 c.c. of distilled water were injected into the peritoneal cavities of four rabbits daily for fourteen days. The rabbits were killed 1, 2, 3 and 4 weeks after the last injection.

Results.—The pathological lesions found in the four rabbits injected intraperitoneally with the recovered phospholipid were all very similar, both in the gross appearance and microscopic sections.

Grossly, the peritoneal surfaces were smooth, moist and glistening except for a few fine adhesions between adjacent loops of bowel and a number of discrete nodules either subserosal or adhering to the surfaces of the liver and spleen and extending into these organs. There was a generalized prominence of the milk-spots. The omentum was thickened and contained a number of rounded, firm, grey-pink granular nodules situated between its peritoneal layers. It was not adherent to other structures in any of the animals. The nodules adhering to the peritoneal surfaces and in the subserosa varied in size from 0.1 to 2 cm. A number of the larger nodules on section showed central necrosis. They contained considerable amounts of thick, yellow caseous material. This material did not resemble the pus usually found in rabbits.

Microscopic sections of the different nodules, stained with hæmatoxylin and eosin, showed pathological changes varying from small clusters of foreign-body giant-cells and monocytes filled with lipoid granules and surrounded by lymphocytes and fibroblasts to dense masses of connective tissue. Fairly typical tubercle formation is seen in many of these foci. The general picture is one of a central aggregation of epithelioid cells, monocytes and foreign body giant-cells filled with lipoid material, interspersed with lymphocytes and surrounded by layers of epithelioid cells and fibroblasts. Langhan's giant-cells in varying numbers are seen in some of these nodules. A number of the larger nodules show varying amounts of amorphous, acidophilic debris. In numerous foci the fibroblasts tend to be arranged in whorls closely resembling the lesions of tuberculosis, silicosis and experimental silicosis.

Conclusions.—The pathological findings were similar to those in the animals treated with particulate silica and comparable to those produced by Sabin with her tuberculo-phospholipids and brain lecithin.

The accompanying photomicrographs are taken from several typical microscopic fields.

COMMENT AND CONCLUSIONS ON EXPERIMENTS I, II AND III

A phospholipid has been extracted from early experimental silicotic lesions. The amount increases in proportion to the extent of the early cellular reaction. This material was also present in very slightly increased amounts in kaolin-produced lesions. This material, when free of

silica and re-injected into the peritoneal cavities of rabbits, produces a fibrotic reaction similar to that produced by the injection of finely particulate quartz. These lesions are also very similar to those produced by Sabin with the phospholipid fractions of tubercle bacilli. The recovered phospholipid, in much smaller amounts than those used by Sabin, produces fairly extensive lesions. It is suggested that the characteristic tissue reactions of silicosis and tuberculosis are foreign-body reactions to phospholipids. This accounts for the identical reac-

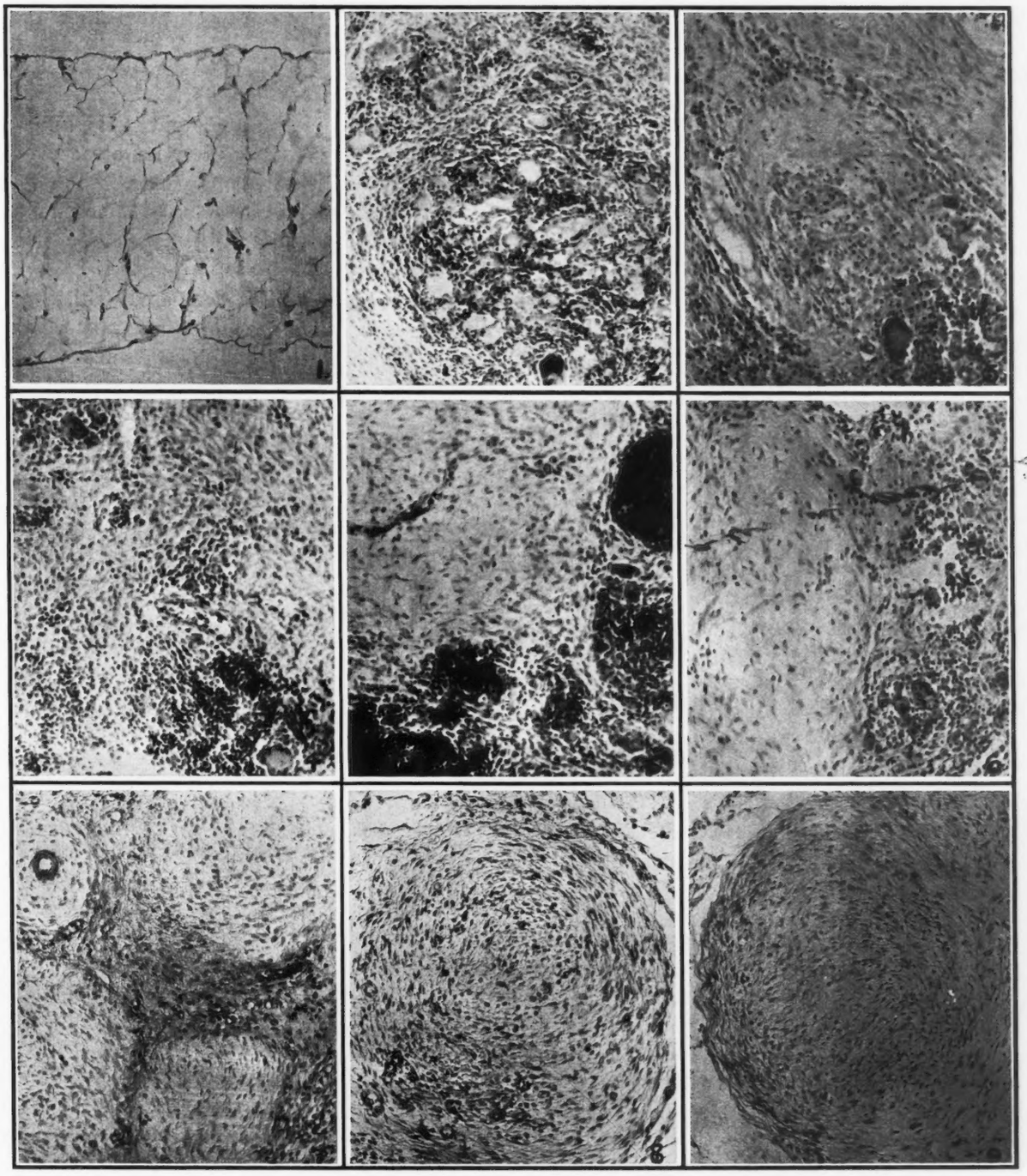


Fig. 1.—Normal rabbit omentum. **Fig. 2.**—Early lesions in rabbit omentum produced by intraperitoneal injection of silica-free phospholipid recovered from the lungs of rabbits with experimental silicosis. The section shows the phospholipid in monocytes and foreign-body giant-cells, lymphocytic infiltration and one Langhan's giant-cell. **Figs. 3, 4, 5 and 6.**—Sections from rabbit omentum showing a tubercle-like reaction consisting of zones of lymphocytes and fibroblasts surrounding phospholipid deposits with occasional endothelial giant-cells. **Figs. 7, 8 and 9.**—Sections of rabbit omentum showing rounded aggregations of fibroblasts which resemble the lesions in experimental silicosis.

tion to inhaled quartz dust and the tubercle bacillus in silicosis and pulmonary tuberculosis.

Silicates, for example, kaolin, stimulate an early monocytic response similar to that of quartz, but the subsequent chemical and histological findings are different. The silicates are taken up by monocytes and foreign-body giant-cells and remain apparently inert in these cells. The kaolin lungs used as a control showed a slightly increased phospholipid content throughout the experiment. This may probably be accounted for by the small increase in number of this type of cell. The reason that the silicates do not produce fibrosis may be that they have no effect, or only a minimal one, upon the metabolic processes of the monocytes.

It is evident from the work of Sabin and others that either the phospholipids of the tubercle bacilli alone, or in combination with the lipoids of numbers of disintegrated monocytes, produce the typical pathological lesions of tuberculosis. Early typical lesions of tuberculosis will disappear when the process is checked. This is not the case in the lesions of silicosis. The difference is probably due to the fact that when the tubercle bacilli are all destroyed the toxic phospholipids are gradually phagocytosed and removed, with the later removal of the scar tissue. In silicosis the monocytes become loaded with quartz particles; there is a physico-chemical change in the monocyte that causes disintegration of the cell and discharge of its contents; the liberated silica, which is practically unchanged, can now stimulate a further monocytic response with further disintegration and so on, indefinitely. The extent of the reaction depends upon the amount of inhaled silica which is imprisoned in the lung.

SUMMARY

1. The amount of phospholipid in the lungs of rabbits increases rapidly after intratracheal injection of finely particulate quartz.

2. The increasing amount of phospholipid corresponds with the type and intensity of the cellular reaction.

3. Re-injection of the recovered (silica-free) phospholipid stimulates a fibrous tissue reaction similar to that produced by the injection of finely particulate quartz.

4. A reason for the similarity of the lesions of silicosis and pulmonary tuberculosis is suggested.

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HISTOLOGICAL VARIATIONS IN FETAL CALVES' THYROIDS AND A COMPARISON WITH MATERNAL THYROIDS*

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IN recent publications,[1, 2] we have demonstrated that the normal thyroid varies a great deal in its histological appearance and that these variations are to be considered normal. Marine originally described the normal thyroid as a mass of rounded acini, varying slightly in size, and with walls formed of uniform low cubical cells with small, darkly-staining nuclei. The cells are arranged in a single layer. The stainable colloid is dense and homogeneous, and, staining uniformly, it sharply abuts upon the epithelial cells. Marine believed that a columnar cell is pathological. Wilson,[3] on the other hand, states that the normal epithelium is almost entirely columnar in type and that the low cuboidal form is usually found only in pathological states. In contradistinction to this we found that the thyroid gland must be given a much wider range of physiological variation, depending on

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age, season, activity, etc. After a careful study of approximately 1,000 glands from a variety of animals from youth to old age we arrived at the following conclusions.

In younger animals hyperplastic active glands are usual[1, 2] and are physiological, not pathological. Small acini, tall columnar cells and little colloid is the characteristic picture. As age advances the acini tend to become larger, varying in size and not in number; colloid increases in amount; cells become cubical and we find a picture more like what is described as the normal gland. At an older age the colloid appears to be laid down in even greater quantity, the cells tend to become flattened, interacinar fibrous tissue increases, and the picture is one of a gland past its prime, sluggish and gradually declining to decay and death.

Following this observation we considered it of importance to trace the histological variations found in intrauterine life. It was also deemed of interest to determine whether any histological relationship exists between the fetal and maternal thyroid.

METHOD OF STUDY

Fetal thyroids were collected from cows at the Canada Packers Abattoir in Winnipeg. In the abattoir each animal was given a number. One of the first things done was the removal of the head and recovery of the thyroid. This gland was immediately placed in a can properly numbered and containing 10 per cent formalin. This can was then taken to where the abdomen was opened, and if the cow was pregnant the fetal thyroid was removed and its age noted. As the glands were placed immediately in formalin no post-mortem changes were possible. All glands were sectioned in routine fashion. The study of each microscopical section was done in the same manner as in our previous work,[1, 2] data being tabulated as to colloid content, vacuolation, cell type, tufting, presence of lymphoid tissue, amount of stroma, vascularity, and in the case of the fetal thyroids the degree of differentiation was carefully noted.

MORPHOGENESIS OF THE HUMAN THYROID

Early embryologists stated that the thyroid takes its origin from the pharyngeal floor between the first two branchial clefts. Closely following the evagination and downward migration, they observed tube-like structures which they interpreted to be primary tubules of the thyroid. They believed the follicles were formed by constrictions of these tubules. They also attributed the increase in number of follicles to budding growths, constriction of follicles and fetal rests.

Edgar H. Norris[4] has done a great deal to clear up the true histology of the thyroid, basing his conclusions upon the study of 72 human embryos, utilizing several sections and reconstructing models from drawings. Arbitrarily, he describes eight stages. The first stage is between the formation of the entodermal pharynx and the appearance of the thyroid *Anlage*. The second is represented by a shallow evagination of the pharyngeal floor which lies between the ventral extremities of the first two pairs of gill pouches. This occurs in the 2 to 4 mm. embryo, and the thyroid pouch is already partially obliterated by epithelial thickening. The third is the early growth stage. The primitive thyroid may appear as an oval or pyriform process suspended from the floor of the mouth. This process may be solid, hollow or bilobed. The elongation of the stalk he describes as the fourth stage, at which time there is beginning separation. The complete separation occurs at different ages, the earliest seen in a 3.9 mm. embryo. Cell boundaries are beginning to be differentiated in the fifth stage. The sixth stage in the differentiation is possibly the reason for the earlier descriptions which mention tubules. In this there is cavity formation in the *Anlage*. These cavities appear to coalesce, but the majority open to the surface and are invaded by the surrounding mesenchyma. After mesenchymal invasion the epithelial cells are still compactly arranged except for the fenestrations. Norris calls this the seventh stage. The eighth, or follicular stage is divided into primary and secondary follicle-stage formation. The primary follicle arises in the fenestrated plate from groups of cells independent of the original cavity lining cells. The plate is gradually replaced with these small follicles, but not by degeneration of the central cells. The formation of secondary follicles may begin in the 65 mm. embryo and continues rapidly. They develop "on the follicle wall and may become separated from the parent follicle while in the solid state, or may develop lumina while connected with the wall of the mother-follicle and subsequently become constricted off". Hollow buds may have a lumen continuous with the mother-follicle and become

constricted off, or the parent follicle may become constricted. The formation of secondary follicles in the embryo is most rapid between 80 and 165 mm., but there is no marked increase in the size of the gland. Few follicles are formed after this, but the size of the gland increases owing to increase in the size of the follicle. This observation by Norris and Wilson is significant when we consider the increase in the basal metabolic rate of the mother during pregnancy.

Our present investigation does not include the early development of the thyroid, but has to do with its development from the second month on, with special reference to follicle development, the appearance of colloid and the relationship of the activity and general structure with that of the mother's thyroid.

Table I shows the histological variations found in 17 fetal calves' thyroids two months of age. All fetal thyroids are marked A2. The mother's thyroids are marked A and were classified first, the corresponding fetal thyroid being classified immediately below. A study of Table I shows that the stage of development at two months varies a great deal as judged by acinar formation, the amount of colloid present, and type of cell found. The type of cell found depends on whether it is in the wall of an acinus or lying free in a mass of ephithelial cells. Without going into a minute cytological description of the cells, which will be dealt with in another paper, it was found that where differentiation into acini had not taken place, the cells were oval or irregular in shape. Scattered throughout the section one sees groups of cells apparently lining up in a loose follicle formation, still retaining their oval shape and their cell margins not in contact. It appeared as if some force gradually rearranged them, first in loose circular fashion, and later packing them tighter until a well-formed acinus was produced, at which time the individual cell had become cubical (Figs. 1 and 2). At no time did we see rods of cells lying in contiguity, gradually separating to form acini as is shown in numerous textbooks on this subject. It appeared rather a loose re-arrangement from a mass of cells lying in solid masses.

TABLE I.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
39A	–	xx	0	Col.	x	0	xx	xx	Hyper. x with fibrosis.
39A2	25%	P.	0	Cub.	0	0	0		25% differentiation.
41A	–	xxx	0	Cub.	0	0	0	0	Normal resting.
41A2	0	0	0	Emb.	0	0	0	0	No differentiation.
51A	–	xx	0	Cub.	0	0	xx	0	Normal active. Fibrosis x.
51A2	occas.	0	0	Emb.	0	0	0	0	Occasional acinus only.
54A	–	x	0	Col.	x	0	xx	xx	Hyper. x.
54A2	0	0	0	Emb.	0	0	0	0	No differentiation.
17A	–	xxx	0	Cub.	0	0	x	0	Normal active.
17A2	occas.	P.	0	Emb.	0	0	0	xxx	Occasional acinus vascular.
18A	–	xx	0	Cub.	0	0	xx	xx	Normal active.
18A2	75%	P.	0	Cub.	0	0	x	xxx	75% differentiation.
38A	–	xx	0	Col.	x	0	xx	x	Normal active fibrosis x.
38A2	25%	P.	xx	Cub.	0	0	0	0	25% differentiation.
45A	–	xx	0	Col.	x	0	xx	xxx	Hyper. x.
45A2	25%	P.	x	Cub.	0	0	0	0	25% differentiation.
49A	–	xxx	x	Cub.	0	0	x	x	Normal active.
49A2	50	P.	xx	Cub.	0	0	xx	0	50% differentiation.
55A	–	xx	0	Cub.	0	0	0	0	Normal resting.
55A2	0	0	0	Emb.	0	0	0	0	No real differentiation.
58A	–	xx	0	Cub.	0	0	0	0	Normal resting.
58A2	25	P.	xx	Cub.	0	0	x	x	25% differentiation well formed.
70A	–	xx	0	Col.	x	x	xxx	x	Hyper. xx.
70A2	0	P.	0	Cub.	0	0	x	0	Many immature acini.
509A	–	xxx	xx	Cub.	0	0	0	0	Normal active with fibrosis.
509A2	75%	xx	0	Cub.	0	0	0	x	Acini large. Much colloid.
502A	–	xx	0	Cub.	0	0	0	0	Normal active.
502A2	0	x	0	Cub. & Col.	0	0	0	xx	Acini beginning.
536A	–	xxxx	0	Cub.	0	0	x	x	Normal resting.
536A2	75%	x	0	Cub.	0	0	0	xx	75% differentiation.
537A	–	xxx	0	Cub.	0	0	0	0	Normal resting.
537A2	50	x	0	Cub.	0	0	0	x	50% well formed.
533A	–	xx	x	Cub.	0	0	x	xx	Normal resting.
533A2	50	xx	0	Cub.	0	0	0	x	60% well formed.

In the 17 glands studied, there was an occasional acinus present in two, 25 per cent acini formation in four, 50 per cent in three, 75 per cent in three and in five glands one could find no evidence of acini whatsoever. Colloid was present in 13 out of 17 glands, including some glands where acinus formation had not yet taken place. In these glands clumps of cells not yet arranged in acinar formation would be seen with colloid-like material distributed amongst them. Vacuolation, which in our opinion is evidence of functional activity was present in four of the fetal thyroids (Fig. 2 and Table I).

TABLE II.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
304A	–	xx	0	Col.	x	0	x	x	Hyper. x.
304A2	50%	x dense	0	Cub.	0	0	0	0	Acini well formed. Small.
73A	–	xx	0	Cub.	0		0	0	Normal resting.
73A2	50	x	0	Cub.	0	0	0	0	50% differentiation.
26A	–	xx	0	Cub.	0	0	xx	xx	Normal resting.
26A2	25	x	0	Cub.	0	0	0	0	25% small acini.
27A	–	xxx	x	Cub.	0	0	0	0	Normal active.
27A2	50	x	xx	Cub.	0	0	0	0	Cells very large. Much vacuolation.
21A	–	xx	0	Col.	0	0	x	x	Hyper. x.
21A2	95%	x	xxx	Col.	0	0	x	x	95% acini. Colloid very vac.
23A	–	xx	0	Cub.	x	x	x	x	Normal resting.
23A2	50	x	xx	Cells like adult	–	–	0	0	Acini more like adult.
40A	–	xx	0	Col.	x	0	x	x	Hyper. x.
40A2	75	xx	x	Cub. & Col.	0	0	0	0	75% well formed. Compact.
523A	0	xx	x	Cub.	0	0	x	0	Normal resting.
523A2	25	x	0	Col. & Cub.	0	0	0	0	Varying degree of differentiation. Small and large acini. Not sharp-defined.
521A	–	xx	0	Cub.	0	0	0	0	Normal resting.
521A2	75	xx	0	Cub.	0	0	0	0	75% differentiation. Uniformation.
524A	–	xxx	x	Cub.	0	0	0	0	Normal resting.
524A2	50	x	0	Cub.	0	0	0	0	50% well formed.
536A	–	xxxx	0	Cub.	0	0	Fib.	x	Normal resting.
536A2	75	x	0	Cub.	0	0	0	xx	75% well formed.
537A	–	xxx	0	Cub.	0	0	0	0	Normal resting.
537A2	50	x	0	Cub.	0	0	0	x	50% well formed.
533A	–	xx	x	Cub.	0	0	x	xx	Normal resting.
533A2	50	x	0	Cub.	0	0	0	x	50% well formed.
532A	–	xx	0	Cub.	0	0	x	x	Normal resting.
532A2	50	x	0	Cub.	0	0	0	xx	50% well formed.
535A	–	xx	0	Cub.	0	0	x	0	Normal resting.
535A2	75	xx	0	Cub.	0	0	0	xx	75% well formed.
538A	–	xxx	x	Cub.	0	0	0	0	Normal resting.
538A2	0	x	0	Emb.	0	0	0	xx	Early immature acini formation.

TABLE III.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
312A	–	xxx	x	Flat	0	0	x	x	Normal resting.
312A2	100%		0	Cub.	0	0	0	0	Normal active. Complete differentiation.
29A	–	xx	0	Col.	0	0	x	x	Hyper. x.
29A2	75%	xx	0	Cub. & Col.	x	0	0	0	Acini well formed. Irregular like in hyper. gland. Many colloid poor Cells tall and col. Well formed. *Hyperplastic.*
573A	–	xxx	0	Cub.	0	0	x	0	Normal resting.
573A2	50	xx	0	Cub.	0	0	0	0	50% differentiation. Well formed.
522A	–	xx	x	Col.	x	0	xx	x	Hyper. x.
522A2	75	xx	0	Cub.	0	0	0	0	75% well formed.
517A	–	xxx	0	Cub.	0	0	x	x	Normal resting.
517A2	90	xx	0	Cub.	0	0	0	x	90% well formed acini. Compact.
501A	–	xx	x	Cub.	0	0	xx	0	Normal resting.
501A2	50	x	0	Cub.	0	0	0	xx	50% very small.
534A	–	xxx	0	Cub.	0	0	Fib.	0	Normal resting.
534A2	50	x	0	Cub.	0	0	0	xx	50% well formed.

TABLE IV.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
500A	–	xx	0	Col.	x	x	x	x	Hyper x.
500A2	75	xx dense C	x	Cub.	0	0	0	0	75% acini. Well formed.
75A	–	xx	0	Col.	xx	x	xxx	xxx	Hyper. xx.
75A2	75	xx	0	Cub.	0	0	0	0	Colloid dark. Acini close together.
7A	–	xx	0	Col.	0	0	x	xx	Hyper. x.
7A2	75	xx	xx	Cub. & Col.	0	0	0	0	75% acini. Vacuolation marked.
11A	–	x	xx	Col.	x	0	xx	xx	Hyper. x.
11A2	90	xx	x	Cub.	0	0		0	Acini and colloid adult type.
37A	–	xxx	0	Cub.	0	0	x	xx	Normal active.
37A2	100%	xxx	x	Col.	x	x	x	x	Cells tall and columnar. Hyper. x disc. tufting.
A	–	xxx	x	Cub.	0	0	x	xx	Normal active.
A2	50%	xx	xx	Cub.	0	0	0	x	50% differentiation.
529A	–	xxxx	x	Flat	0	0	–	–	Colloid xx.
529A2	75	xx	0	Cub.	0	0	0	x	75% well formed.

In comparing the histological picture found in the maternal and fetal thyroids in the series we could find no evidence of any relationship. In two the mother's thyroid was classified as normal resting glands, normal active in one, and hyperplastic in the fourth. In only one of the adult glands was vacuolation present. It is of interest to note, however, that in the four adult thyroids that were hyperplastic, the corresponding fetal thyroids in two were undifferentiated and only 25 per cent differentiated in the remaining two.

In the group of 16 fetal and maternal thyroids studied at the third month of gestation, differentiation was much more advanced (Table II). Only one fetal thyroid showed no differentiation. Two were 25 per cent differentiated, eight 50 per cent, four 75 per cent, and one 95 per cent or almost completely differentiated. Colloid was present in all glands, and in three cases was classified as xx, which is the normal amount based on our adult classification.

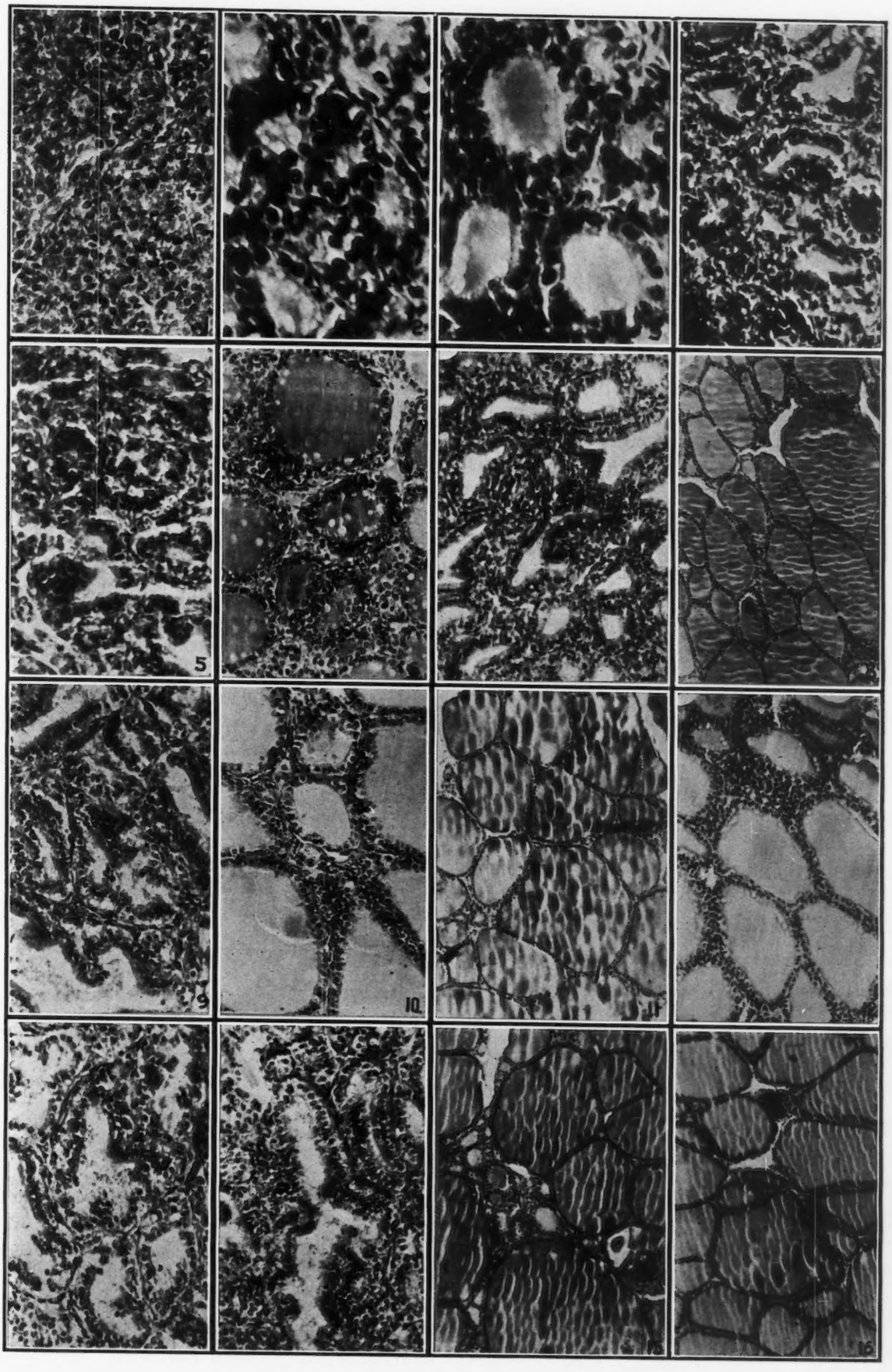
Vacuolation was present in 25 per cent and in gland No. 21A2 was graded xx (Fig. 3). In this gland differentiation was 95 per cent completed and the cells were columnar. The cells were cubical in 12 cases, columnar in 3 and still embryonic in type in 1 undifferentiated gland. No relation between fetal and maternal thyroid could be seen in this group.

Tables III and IV show the classification of the thyroids of four and five month fetuses and of the thyroids of the corresponding mothers.

In the fourth month seven fetal and maternal thyroids were examined. In this group no fetal thyroid was found to be less than 50 per cent differentiated. In 2 differentiation was complete, 2 were 75 per cent differentiated and 3 were 95 per cent. Colloid again was present in all and vacuolation was absent in all.

See illustrations on right-hand page.

Fig. 1.—Two months' fetal thyroid, No. 55A2, x 400. A mass of epithelial cells with beginning early acinar formation. Note the loose arrangement of the cells in the newly-formed acini. **Fig. 2.**—Two months' fetal thyroid, No. 49A2, x 700. Well marked acinar formation and vacuolation, indicating functional activity of the fetal thyroid as early as the second month. **Fig. 3.**—Three months' fetal thyroid, No. 21A2, x 700. Note well-formed acini lined by cubical and columnar cells; colloid two plus, and vacuolation two plus. **Fig. 4.**—Four months' fetal thyroid, No. 29A2, x 350. Hyperplastic gland showing well formed acini with tall columnar cells, tufting, colloid two plus, and in a gland not completely differentiated. **Fig. 5.**—Fifth month fetus, No. 37A2, x 350. Hyperplastic gland characterized by decrease in colloid, tall columnar cells, tufting, and moderate vascularity. **Fig. 6.**—Six months' fetal thyroid, No. 67A2, x 200. Early hyperplasia characterized by well marked vacuolation, colloid two plus, low columnar cells, interacinar tissue and vascularity, two plus. **Fig. 7.**—Six months' fetal thyroid, No. 9A2, x 200. Typical of Graves' disease. Note the lack of colloid, tufting, and tall columnar cells. **Fig. 8.**—Maternal thyroid corresponding to Fig. 7, No. 9A, x 100; normal thyroid gland. **Fig. 9.**—Seven months' fetal thyroid, No. 65A2, x 200; markedly hyperplastic gland. Note the tall columnar cells and tufting. Compare with Fig. 10 (mother's thyroid). **Fig. 10.**—No. 65A, x 200; normal active thyroid gland. **Fig. 11.**—Eight months' fetal thyroid, No. 525A, x 200; normal resting. **Fig. 12.**—Eight months' fetal thyroid, No. 525A2, x 200. A normal active gland bordering on early hyperplasia. **Fig. 13.**—Maternal thyroid, No. 508A, x 200. A hyperplastic thyroid gland characterized by loss of colloid, columnar cells and tufting. (Compare this with the corresponding fetal thyroid, Fig. 14). **Fig. 14.**—Eight months' fetal thyroid, No. 508A, x 200. Hyperplasia four plus. Note the marked tufting and tall columnar cells. **Fig. 15.**—Maternal thyroid, No. 518A, x 100. Colloid goitre one plus. **Fig. 16.**—Eight months' fetal thyroid, No. 518A2, x 100. A normal active gland. It is to be noted that the figures expressing the magnification are, on account of the reduction, to be regarded as comparative and not absolute.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Fetal thyroid 29A2 was of special interest as it was the first one found to be definitely hyperplastic (Fig. 4). This gland was not completely differentiated, approximately 75 per cent of the cells being arranged in acinar formation. The lining cells were cubical and columnar, and the acini which were well formed were irregular and tufted as in the adult hyperplastic gland. It is of interest to note that the mother's gland was also hyperplastic.

Seven pairs of glands were studied in the fifth month of pregnancy (Table IV). In the 7 fetal thyroids studied one was 50 per cent differentiated, four 75 per cent differentiated, one 90 per cent and one 100 per cent differentiated. Colloid was present in all and the cells were cubical in 5 and columnar in 2. Vacuolation was present in a much higher percentage than heretofore, being present in 5 out of the 7. Gland 37A2 was of interest as vacuolation was present, the cells were columnar, tufting was present, and the gland was classified as hyperplastic (Fig. 5). The mother's thyroid was classified as normal active. Fetal thyroid 7A2 showed very well marked vacuolation but was not considered hyperplastic.

In the last four groups, the fetal thyroids very closely simulated the adult type, except for the smaller acini present. Differentiation in all was almost complete and hyperplasia in the fetal thyroid not uncommon.

Ten fetal and 10 adult thyroids were examined at the stage of six months' gestation (Table V). The fetal thyroids were all at least 90 per cent differentiated. Colloid was present in all fetal thyroids and 90 per cent showed well marked vacuolation. Very definite tufting was present in 3 cases and in 1 case (67A2) vacuolation was graded xxx (Fig. 6). In this group we found two very interesting fetal thyroids. Gland No. 9A2, 100 per cent differentiated, vacuolation xxx, tufting two plus, simulated a typical Graves' disease (Fig. 7). According to our classification in 1931,[6] we would have considered the gland pathological. The mother's thyroid was normal (Fig. 8). Fetal thyroid No. 67A2 is of interest on account of the tremendous vacuolation (Fig. 6). A careful study reveals no definite relationship between the activity of the mother's thyroid and that of the fetus up to this stage.

Owing to the small number of glands in the 7th and 8th and 9th month group the last three groups will be considered together. Eleven fetal thyroids and the corresponding mother's thyroids were examined. An examination of the mother's thyroids showed a wide range of variation, 1 being classified as colloid, 3 as normal, 4 normal active, 1 hyperplastic, 1 hyperplastic xx, 1 hyperplastic xxx, and 1 hyperplastic xxxx. The fetal thyroids also varied a great deal. None, however, were classified as colloid and one only as normal resting.

Six glands were classified as normal active, one hyperplastic one plus, two hyperplastic three plus, and one hyperplastic four plus. A

TABLE V.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
12A	–	xxx	0	Cub.	0	0	xx	0	Normal resting and fibrosis xxx.
12A2	90%	xxx	xx	Cub.	0	0	x	x	Colloid denser.
1A	–	xx	0	Cub.	0	0	x	x	Normal active.
1A2	90	x	0	Cub.	0	0	0	0	Hyper. x. Very disc. vacuolation.
9A		xx	0	Cub.	0	0	xx	0	Normal active.
9A2	100%	x	xxx	Col.	xx	xx	0	0	Typical Graves.
10A	–	xx	0	Cub.	0	0	x	0	Normal active.
10A2	90	xx	x	Cub. & Col.	x	x	x	x	Hyper. x.
15A	–	xx	0	Cub.	0	0	x	x	Normal active.
15A2	90%	xx	xx	Cub.	0	0	x	x	90% well formed. Normal active.
66A	–	xxx	xx	Cub.	0	0	x	xxx	Normal active.
66A2	100%	x	x	Col.	xx	x	xx	xx	Hyper. x.
67A	–	xxxx	x	Cub.	0	0	0	x	Normal resting.
67A2	95%	xx	xxx	Col.	x	0	xx	xx	Tremendous vacuolation. Hyper. x.
506A	–	xxxx	0	Flat	0	0	0	0	Colloid x.
506A2	90	xx	xx	Col.	xx	0	0	xx	Hyperplasia.
541A	–	xxx	0	Cub.	0	0	x	xx	Normal active.
541A2	90%	xx	xx	Col.	x	0	0	0	90% hyperplasia.
510A									No section.
510A2	90	xx	x	Col.	0	0	0	x	90% differentiation. Normal active.

hyperplastic gland in a seven month fetus, however, did not of necessity mean that we would find a hyperplastic thyroid in the mother. Numbers 65A and 65A2 are very good examples of this, for although the fetal thyroid is markedly hyperplastic (Fig. 9) that of the mother's was found to be normal active (Fig. 10). (See Table VI).

In the 8th and 9th months (Tables VII and VIII) however, it would appear that some striking change has taken place, bringing about a very close relationship between fetal and mother's thyroid. A study of the 8th and 9th month table is interesting from this point of view, as in almost every case there appears to be a very close similarity in histological structure. In animals Nos. 525A and 525A2 the mother's thyroid was classified as normal active. Fig. 12 shows slight increase in the fetal thyroid activity, which is what one would naturally expect. Group 508A and 508A2 shows the mother's thyroid to be hyperplastic xxx (Fig. 13), while the fetal thyroid was classified as hyperplastic xxxx (Fig. 14). In only one group of glands, 518A and 518A2, was there a spread of more than one grade. In this pair of glands the mother's thyroid was graded colloid (Fig. 15), and the fetal thyroid normal active, which in itself is not a very large degree of difference (Fig. 16).

DISCUSSION

Cooper[5] traced the histology of the thyroid in human fetuses a few years ago on a limited number of subjects, and Abbott and Ball[6] reported on a histological survey of 100 human fetuses from the 6th to the 9th month in 1931. The present survey is based on animal tissue entirely and differs considerably from their findings. In the case of the work of Abbott and Ball, the authors at that time did not appreciate the wide range of histological varia-

TABLE VI.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
65A	–	xxx	0	Cub.	0	0	0	xx	Normal active.
65A2	100%	0	0	Col.	xx	x	x	x	Hyper. xxx. Marked tufting.
507A	–	xx	xxxx	Col.	xx	x	xx	xx	Hyper. x.
507A2	100%	x	xx	Col.	xx	x	x	x	Hyper. x.

TABLE VII.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
314A	–	xx	0	Cub.	0	0	x	x	Normal active.
314A2	75%	xx	0	Cub.	0	0	0	0	75% differentiation. Normal active.
516A	–	xx	xx	Cub.	0	0	x	x	Normal active.
516A2	100	xx	x	Cub.	0	0	xx	xx	Normal active (adult).
518A	–	xxxx	x	Flat	0	0	x	xx	Colloid x.
518A2	100	xx	x	Cub.	0	0	x	x	Normal active.
525A	–	xxx	x	Cub.	0	0	x	x	Normal resting.
525A2	100	xx	xx	Cub.	0	0	0	x	Normal active.
508A	–	x	xx	Col.	xxx	xxx	x	xx	Hyper. xxx.
508A2	100	x	xxxx	Col.	xxxx	xxxx	0	x	Hyper. xxxx.
527A	–	xxx	0	Cub.	0	0	0	0	Normal resting.
527A2	100	xx	x	Cub.	0	0	0	0	Normal resting.
528A	–	xx	x	Cub.	0	0	x	xx	Normal active.
528A2	100	xx	xx	Cub.	0	0	x	xx	Normal active.

TABLE VIII.

Animal No.	*Acini present*	*Colloid*	*Vacuolation*	*Cells*	*Hyperplasia*	*Tufting*	*Stroma*	*Vessels*	*Classification and remarks*
					x				
313A	–	xx	0	Col.	xxx	x	xx	xx	Hyper. xx.
313A2	100	0	0	Col.	x	x	xxx	xxx	Hyper. xxx.
20A	–	xxx	0	Cub.	0	0	x	0	Normal resting.
20A2	100	xx	x	Col.	x	0	x	0	Normal resting. Acini adult.

tion one finds in the thyroid, and what they considered pathological at that time is now considered a normal physiological phase in thyroid activity.

The 2nd month fetal thyroids which Cooper found consisted of a mass of small round epithelial cells with large, deeply stained nuclei. The cells at the periphery of the gland were much more compact than in the centre of the gland where they are loosely arranged. No colloid, follicles or vacuolation were present. In contradistinction to this we found 70 per cent of our 2nd month fetal thyroids with well formed acini, 76 per cent contained colloid, and 4 glands or 23 per cent, showed vacuolation. This is definite evidence that thyroid differentiation takes place earlier in calves than in human fetuses; as vacuolation in our opinion indicates thyroid function, it would appear that we here have definite proof that the thyroid functions in the fetus as early as the second month of intrauterine life.

Ian Murray[7] has shown that vacuolation will appear in the periphery of the acini in thyroids which have not been fixed when kept at room temperature for a few days. As all our tissues were fixed within an hour after death, any vacuolation present was considered definitely due to physiological absorption and not to post-mortem changes. From the second month on we find a rapid increase of differention up to the sixth month, when 80 to 90 per cent of the fetal thyroids are completely differentiated. In the third and fourth month Cooper describes tubules of cells which can be seen branching and budding. In all our work, in spite of diligent search, we have never been able to prove to our own satisfaction that this process occurs.

In the fifth month Cooper first found colloid and acini. As we have pointed out previously both are found in over 50 per cent of our sections in animal thyroids as early as the second month of intrauterine life. Elkes states colloid appears in the human from 4½ to 6½ months. Miller, Peremeschko and Boechat say it appears at the fifth month. On the other hand Wolfer states colloid formation does not commence until the end of fetal life or even until after birth. These observations do not hold true as far as the development of the fetal thyroid in calves is concerned.

Vacuolation is found according to Cooper in the 7th month of intrauterine life. In our series of animal thyroids we found it present in 23 per cent of glands at the second month of intrauterine life.

Abbott and Ball[6] in their work on fetal thyroids found evidence of hyperplasia in the latter part of intrauterine life. In our present work we found definitely hyperplastic thyroid glands in the fourth month of intrauterine life.

In our second problem the fetal and maternal thyroids were compared in an endeavour to trace any histological similarity at monthly periods during intrauterine life. From the 2nd to the 7th month no similarity whatsoever could be detected. In the last two months of fetal life, however, a striking similarity was found in the nine sets of fetal and maternal thyroids. In only one set of glands was there more than one degree difference in histological classification. In this case the maternal thyroid was classified as colloid x and the fetal thyroid was classified as normal active. A study of Tables VII and VIII together with Figs. 9 to 16 will illustrate the striking similarity between the maternal and fetal thyroids during the eighth to ninth month of fetal life. We appreciate the fact that this work is based upon a small number of animals, and further work is being done to clarify the problem.

SUMMARY

1. Fetal thyroids from calves were studied histologically from the second to the ninth month of intrauterine life. These were then compared with the maternal thyroid in each case.

2. Colloid, vesicle formation, and vacuolation were found as early as the second month of intrauterine life. This is much earlier than has been reported as occurring in human fetal thyroids.

3. Hyperplasia was found in the fourth month of intrauterine life, characterized by tall columnar cells.

4. Differentiation is almost complete by the end of the sixth month of intrauterine life.

5. At the end of intrauterine life the histological appearance is identical with that of the adult except for the fact that the cells are taller, the acini are smaller, and the gland is more vascular.

6. A comparison of the maternal and fetal thyroids shows no similarity for the first seven months. In the last months of pregnancy, however, the similarity in the histological appearances of the fetal and maternal thyroid is striking. This observation, however, is based on a small number of cases and requires further study.

This work was made possible by a grant from the Banting Research Foundation, whose aid we acknowledge with thanks. We also wish to acknowledge their aid for our last publication on "The effect of pregnancy on the thyroid gland", published in the June, 1936, issue of the *Journal*, which we inadvertently failed to do at the time.

The histological sections for this work were prepared by Mr. Teale in the Department of Pathology of the Winnipeg General Hospital under the supervision of Prof. William Boyd. Miss Nason prepared all the micro-photographs for this and all previous publications.

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THE PRODUCTION OF TUMOUR AND TUMOUR-LIKE GROWTHS IN RATS

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Chicago

THE production of tumours in rats was attempted on the basis of the factors shown in a preceding paper* to be characteristic of a strain of mice with a tumour history. The low oxygen consumption of their excised tissue, the low hæmoglobin of their blood, their low red cell count, and their low red cell volume all pointed in the direction of an oxygen deficiency of the cells. The high calcium content of their soft tissues seemed to indicate an excess of base. Although these conditions were compatible with tumour, it was not shown whether they were the cause or the result of tumour. Therefore an attempt was made to bring about oxygen deficiency and excess of base in certain tissues of the rat in order to see whether tumours would result.

As to oxygen deficiency, it has been shown by Barcroft *et al.*[1] that the oxygen tension of the blood which supplies the rabbit fetus is very low, ranging from 20 to 30 mm. of mercury. From this it is apparent that the fetal cells subsist and multiply on a much smaller oxygen consumption than is required by the cells in post-uterine life. From the work of Warburg[2] and others it would seem that excised tumour and embryonic tissue have somewhat the same type of respiration. It would seem probable, therefore, that if in post-uterine life the cells were subjected to the same low oxygen tension as existed in uterine life they might be able to revert to the fetal type of respiration, but in so doing run the danger of becoming tumour cells.

As to excess of base in the tissues, it has been shown by Bálint and Weiss[3] that alkalosis, irrespective of its cause, always leads to an increase of the proliferative process. Thus they found that alkalinity promoted growth of seedlings, healing of wounds, assimilation process of inflammation, tissue-culture growth, and growth of carcinoma. Others[4] have found more potassium in young, actively growing tumours than in slowly growing or old tumours. Roffo,[5] in particular, found that the potassium content of rat tumours was about twice that of the rest of the rat, and was about the same as that of rat embryo. It would seem probable, therefore, that excess of base in the tissues might furnish the predisposing condition for cells that have been forced into the fetal type of respiration to proliferate, and, under favourable conditions, to become cancerous.

The apparent oxygen deficiency and excess of calcium in the tissues of the tumour-strain mice of the preceding study seemed to be part of a general condition. If the same factors occurred in man it was thought they would more likely be part of a local rather than a general condition. Since the significance to the

* See *Canad. M. Ass. J.*, 1937, **36**: 27.

human situation was the foremost consideration in this study, an attempt was made to produce these conditions locally. The uterus was the organ chosen for this purpose, partly because of its comparatively greater isolation, and partly because one horn might serve as a control for any condition produced in the other horn. Oxygen deficiency was brought about by ligating the artery supplying either horn of the uterus. This would probably result not only in deficiency of oxygen but also of substrate to be oxidized, for the ovary as well as the uterus. Nothing was done to bring more base into the tissues other than the injury they would suffer through malnutrition. It was thought that this injury would result in the feeble circulation and retrogressive changes that Wells[6] says are always conducive to calcification. Such a deposition of calcium was counted upon to take place in the tissues affected and to furnish the base thought necessary for the initiation of cell proliferation.

The rats used in these experiments were mostly the common white strain, but also included some descendants of a cross between the white and a hardier black and white strain. This stock had been bred and raised in our laboratories for 7 years, during which time no case of spontaneous tumour was ever observed. Two series of rats were studied. The first consisted of 12 which had the artery to one or both horns ligated near the bifurcation of the uterus, and the second, of 12 litter mates kept as controls. The horn with the unligated blood supply also served as a control. All but two of the rats were kept in small cages to prevent much exercise and to minimize its effect upon the circulation of the blood and the lymph. The remaining two were kept in a large cage which permitted plenty of exercise.

About three months after the operations on the first series, Rat 1, having the left artery ligated, died at the age of 18 months, having on the left side the uterus and ovary growth shown in Fig. 1. This growth had the appearance of a tumour, but in the microscopic section shown in Fig. 2 its cells appear to be more characteristic of inflammatory than tumour growth. The death of this rat seemed to be due to intestinal obstruction brought about not only by the size of the growth but by its absorption of liquid from the intestinal tract.

About 5 months after the operation, which in this case involved the ligation of the arteries to both horns, Rat 2 at the age of 24 months developed the 10 g. growth on the breast shown in Fig. 3. The microscopic section shown in Fig. 4 indicates that this growth was an adenocarcinoma of the mammary gland. This rat died under anæsthesia while the tumour was being removed.

About 6 months after the operation, also involving the ligation of both arteries, Rat 3, a litter mate of Rat 2, also developed the 15 g. breast tumour shown in Fig. 5. The microscopic section shown in Fig. 6 indicates that this growth was an adenofibroma of the mammary gland. This tumour was removed, but about five months later appeared a little lower down another tumour (shown in Fig. 7), of which microscopic sections indicated that it also was an adenofibroma of the mammary gland. Death intervened before the second tumour reached the size of the first. Autopsy did not reveal any pathological conditions other than the tumour and the ligated arteries.

Both of the rats allowed more freedom in a larger cage also developed breast tumours, but not until after a lapse of about twice the time taken by those kept in the smaller cages. One of these, Rat 4, having both arteries ligated when about 16 months old, had 12 months later the breast tumour, weighing 96 g., shown in Fig. 8, microscopic sections of which indicated that it was an adenofibroma of the mammary gland. This rat died under anæsthesia while the tumour was being removed. The other, Rat 5, having both arteries ligated when about 6 months old, had 12 months later the breast tumour shown in Fig. 9. This rat was still living at the time of writing.*

The other 7 rats were sacrificed at intervals of 4 to 8 months after the operation, at ages ranging from a year to a year and a half. Five of these had developed abnormal abdominal growths, the most common being the cyst-like growths. One had set up an accessory circulation by forming adhesions between the uterus and intestines. One had succumbed in the apparent effort to compensate by absorption of liquid from the intestinal tract. This rat pre-

* This rat has since died, having an adenofibroma of the mammary gland, weighing 201 g., or 23 g. more than the rest of the body.

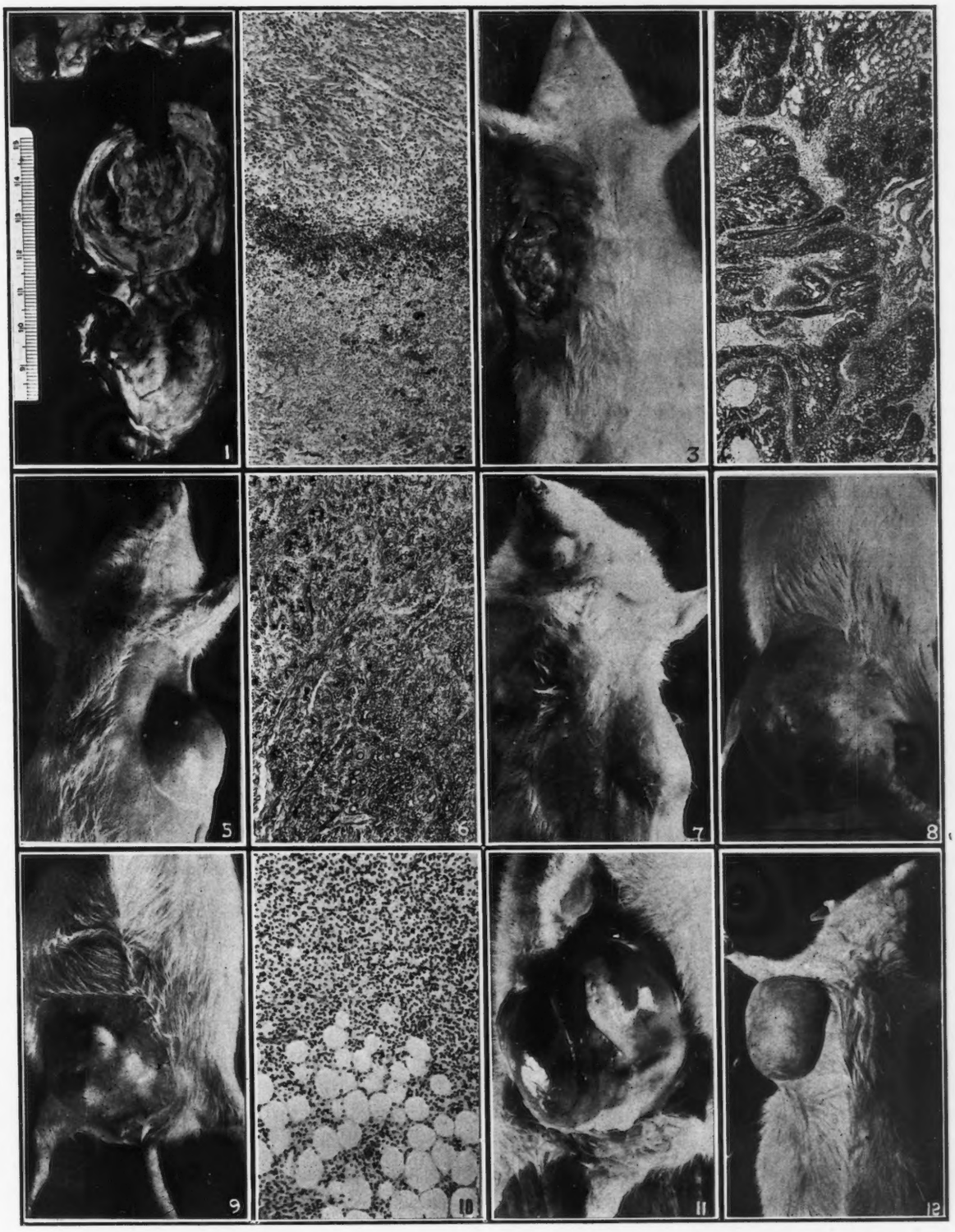

Fig. 1.—Abscess in Rat 1, Series I. **Fig. 2.**—Photomicrograph (x 140) of abscess in Rat 1, Series I. **Fig. 3.**—Adenocarcinoma of Rat 2, Series I. **Fig. 4.**—Photomicrograph (x 45) of adenocarcinoma of Rat 2, Series I. **Fig. 5.**—First adenofibroma of Rat 3, Series I. **Fig. 6.**—Photomicrograph (x 45) of adenofibroma of Rat 3, Series I. **Fig. 7.**—Second adenofibroma of Rat 3, Series I. **Fig. 8**—Adenofibroma of Rat 4, Series I. **Fig. 9.**—Breast tumour of Rat 5, Series I. **Fig. 10.**—Photomicrograph (x 140) of sarcoma of Rat 3, Series II. **Fig. 11.**—Sarcoma of Rat 3, Series II. **Fig. 12.**—Breast tumour of Rat 5, Series II.

sented three nodular growths in the broad ligament with intestinal obstruction. Apparently the other six rats either were examined too soon or were better able to fight off the effects of ligation.

Since during the development of these tumours none of the 12 litter-mate controls exhibited any sign of tumour growth, 6 of these controls were also subjected to the ligation of both arteries. The 6 rats operated on, with the remaining 6 controls, constituted the second series. About 3 months after the operations on the second series Rat 1 of this series died of gangrene, and Rat 2 of intestinal obstruction similar to that already observed in the first series. About the same time Rat 3 died at the age of 19 months, having the abdominal growth shown in Fig. 11. This growth involved the uterus, ovary, and broad ligament, and completely enclosed the caudal portion of the bowel. The microscopic section shown in Fig. 10 indicates that this was a neoplasm, probably a sarcoma. About 6 months after the operation Rat 4 died at the age of 30 months, having in the ligated areas many nodular growths. Rat 5 at the age of about 30 months developed the breast growth shown in Fig. 12. This rat and Rat 6, which was without external sign of tumour, were both living at the time of writing.*

That an oxygen deficiency occurred as a result of these operations was shown by metabolic determinations made on slices of excised uterus by the Warburg differential method.[7] It was found that the oxygen consumption of the horn whose blood supply had been obstructed was less and its glycolysis greater than that of the other horn or of the horns of its litter mate not operated on. This, it should be noted, is the type of respiration characteristic of tumour and of the fetus.

Whether the initiation of tissue proliferation resulted from a deposition of calcium could not be easily determined. However, it seemed probable that the tumour-type of metabolism observed indicated degenerative changes such as Wells[6] found favourable for calcification. In tissues with this type of metabolism Warburg[2] had observed the elimination of much ammonia, which would also serve to increase the base in these tissues. No increase was observed in the pH of the blood.

These conditions of malnutrition and supposed consequent deposition of base were probably responsible for the inflammatory growth of the uterus and ovary of Rat 1 of the first series. Probably the rapidity of the growth in an otherwise healthy rat was responsible for the inflammatory character of the tissues. The extreme measures used apparently brought about growth so quickly that the rat did not live long enough for the tissue to become anything but inflammatory. If the measures adopted had been less extreme, so that growth had taken place very slowly with the rat becoming less healthy, then it is probable that the tissue would have been more tumour-like. That such a transition may take place is supported by Ewing, MacCallum and most pathologists. Some authors[8] have gone so far as to maintain that there is only a difference in degree between the healing of wounds, inflammatory hyperplasia, and tumour-formation. Ewing[9] says,

> A process beginning as a simple inflammatory hyperplasia may in the same individual gradually assume neoplastic qualities. In the thyroid gland of goitre, especially in fish, in the prostate gland of hypertrophic prostatitis, in the uterine mucosa of a glandular endometritis, in the mammary gland of chronic mastitis, and in the lymph nodes of Hodgkin's granuloma are occasionally seen transformations of a functional or inflammatory hyperplasia into a more or less typical neoplasm. . . . . All these considerations strengthen the view that the precancerous condition is of wide occurrence and of much theoretical and practical importance in oncology. In this condition one finds tissues and cells in a state of overgrowth intermediate between inflammatory and neoplastic hyperplasia, exhibiting certain tumour characters which must be judged from different standards for each tissue, and which experience shows are often followed by genuine and usually malignant tumours.

That somewhat different conditions might affect the nature of the growth was shown in the case of Rat 3 of the second series. In this 19-month-old rat the ligation of both arteries had resulted in a growth that enclosed the caudal portion of the bowel without causing bowel obstruction. In the other 18-month-old rat the ligation of one artery resulted in a growth that caused intestinal obstruction. Perhaps the different conditions to be met and, perhaps more important, the different individual health and resistance characteristics with which to meet the different conditions may have accounted for the growth in the latter case being an abscess, while in the former it was a neoplasm.

* These rats have since died, No. 5 having an adenofibroma of the mammary gland, and No. 6, small uterine growths.

That injury to the uterus and ovary should have resulted in tumour of the mammary gland may not appear at first sight consistent with the assumptions that have been made. That a relationship exists between the uterus and ovary, on the one hand, and the mammary gland, on the other, is evidenced by the following facts quoted from Dr. Turner:[10]

1. From the time of puberty there are changes of a cyclic nature taking place in the mammary gland of the female correlated with the ovarian cycle.
2. During early pregnancy the growth and proliferation of the gland is greatly increased, while toward the end of pregnancy the growth phase is gradually superseded by secretory activity which becomes intense about the time of parturition.
3. With the cessation of secretory activity the alveoli and ducts shrink in size, resulting in an enormous decrease in the size of the entire gland. Further growth and renewed secretion normally take place only with another cycle of reproduction.

These relationships between the uterus and the mammary glands were formerly explained on the basis of direct nervous connection, more recently on the basis of hormonal rather than nervous connection. Whatever the explanation, there can be no doubt of the relationship and of the probability that injury to the uterus and ovary might affect the mammary gland and *vice versa.*

The evidence in support of this probability in the case of women has been examined by Taylor, of the Memorial Hospital, New York City. He has studied the etiology of 66 cases of chronic mastitis,[11] and of 217 cases of neoplasms of the breast.[12] From an etiological point of view he considers that tumours of the breast, including carcinoma, are closely related to those of the endometrium, thyroid and ovary, and somewhat also to myomas of the uterus and tumours of the prostate. It will be noted that these are the same tissues referred to by Ewing as being often found in a state of overgrowth intermediate between inflammatory and neoplastic hyperplasia. Taylor found chronic mastitis and neoplasms of the breast often correlated with pelvic lesions, particularly of the uterus and ovaries. He concluded that a functional disturbance of the physiological relationship between the ovary and the breast was an important factor in the genesis of many forms of tumour of the breast. He was able to assemble a lengthy bibliography in support of his observations and conclusions.

SUMMARY

This study is so far too limited to permit definite conclusions on most of the issues involved. It is desired, therefore, only to summarize the facts and leave their explanation to future study. The facts are as follows.

1. In the 18 rats operated on there developed 1 adenocarcinoma, 3 adenofibromas, 1 sarcoma, and 2 mammary gland growths, probably neoplastic, in rats still living.

2. In 5 of the rats operated on, sacrificed in order to make early observations, cyst-like growths were found which might have become tumours or caused tumour development elsewhere if the rats had been allowed to live longer.

3. These tumours and tumour-like growths did not occur elsewhere than on the parts directly affected or closely related.

4. None of the litter-mate controls developed any similar growths, and no tumour has ever occurred spontaneously in the colony of which these rats formed a part.

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"Whatever your career may be, do not let yourselves become tainted by a deprecating and barren scepticism; do not let yourselves be discouraged by the sadness of certain hours which pass over nations. Live in the serene peace of laboratories and libraries. Say to yourselves first: 'What have I done for my instruction?', and, as you gradually advance, 'What have I done for my country?', until the time comes when you may have the immense happiness of thinking that you have contributed in some way to the progress and to the good of humanity. But, whether our efforts are or are not favoured by life, let us be able to say, when we come near the Great Goal, 'I have done what I could.' "—*Pasteur.*

"CASCADE STOMACH" ASSOCIATED WITH IMPAIRED ŒSOPHAGEAL EMPTYING IN A CASE OF "NERVOUS INDIGESTION"*

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ATTENTION has been directed frequently to the psychological factors that form the basis of the gastro-intestinal neurosis. On the other hand, relatively little reference has been made to the physiological mechanisms that determine the actual physical symptoms. Due to belief in the functional nature of the illness, x-ray or other investigation is usually omitted, or its findings overlooked. For example, although "cascade stomach" has been described for over two decades by radiologists, with much speculation regarding its pathological basis, nevertheless correlation with the clinical manifestations has been exceedingly meagre.

The case here reported illustrates an instance of "nervous indigestion", arising clearly from a psychological basis, in which gas-belching and epigastric discomfort were associated with cascade distortion of the stomach and interference with œsophageal emptying. Immediate relief followed treatment, one important detail of which was derived from the findings of fluoroscopy.

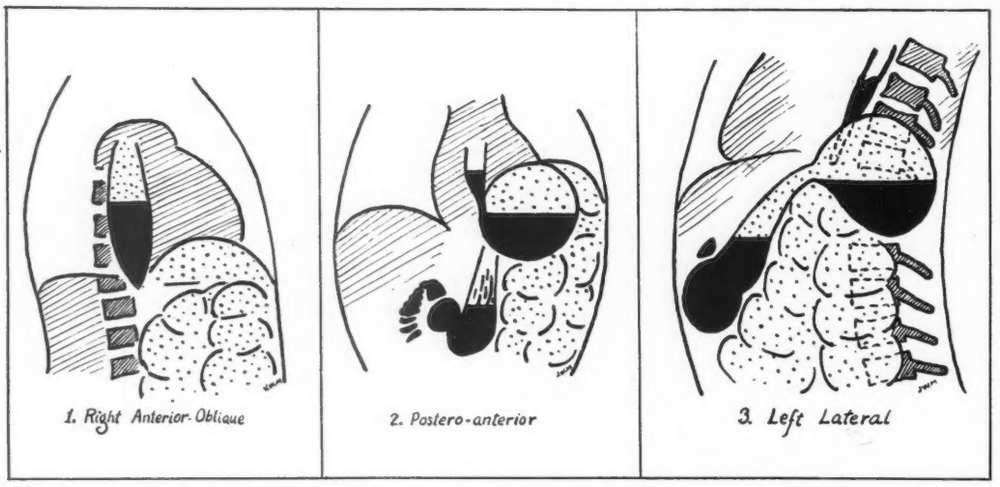

Fig. 1.—Right anterior-oblique view showing left diaphragm elevated by dilated fundus and left colon. The œsophagus, temporarily obstructed, is distended by barium and air. **Fig. 2.**—Postero-anterior view of cascade stomach. **Fig. 3.**—Left lateral view illustrates the upward thrust of the distended colon in producing cascade stomach.

CASE REPORT

D.N., a 28 year old male, referred by Dr. O. W. Bradley, of Verdun, had complained for six weeks of incessant gas-belching, palpitation, and abdominal fullness. He also had smothering feelings and such substernal discomfort during and after eating that meals were reduced to the point of his losing over ten pounds in weight. Nausea and abdominal pain were absent, but he had an inordinate fear of the chest sensations and of the belching, which often rumbled on into the night; he believed his heart was dangerously affected. Sleep, once achieved, was sound, and in the mornings he usually felt well until the belching returned upon the thought of eating. Constipation and flatulence appeared early, but daily catharsis with Epsom salts seemed only to increase the gas.

The diagnosis of neurosis was evident from the history, his symptoms having appeared suddenly after the loss of his job, when his family seemed faced with ruin. The fear of heart disease added to his desperate outlook. Physical examination revealed a hyposthenic habitus, moderate dorso-lumbar lordosis, marked abdominal tympany which blended posteriorly with the resonance of the left lung, and obliteration of the boundaries of Traube's space. Throughout the interview the patient belched constantly, and at intervals emitted a loud sucking noise as air was aspirated. He also swallowed frequently, although the dryness of the mouth suggested diminished rather than excessive salivation.

Fluoroscopy showed the left leaf of the diaphragm to be elevated three inches above the level of its fellow, and relatively immobile during ordinary respirations. The heart was displaced about an inch to the right. The cause of the upward bulge of the diaphragm was obviously an enormous collection of gas in the upper abdomen, much of which, according to haustral markings, lay in the grossly distended limbs of the splenic flexure (Fig. 1).

The first swallow of barium revealed the œsophagus to be elliptically distended with air. There was no disposition to empty until the column of barium had reached a height of 5 to 6 inches above the cardia. Before this, however, the patient complained of substernal discomfort, which he attempted to dispel by

* Read before the Montreal Medico-Chirurgical Society on November 6, 1936.

forcibly swallowing air. This served only to distend further the œsophagus, and frequently air escaped with a loud belch. Eventually, however, after taking more barium and large gulps of air, the œsophagus attained its maximum distension, as shown in Fig. 1. Palpitation and smothering ensued and the patient began to breathe deeply, in a rapid, nervous fashion. Then, it appeared that with each descent of the hitherto inactive left half of the diaphragm barium spurted into the stomach. The process continued until the œsophagus was empty, whereupon the patient resumed normal respirations, and declared that he felt better. Using this cue, it was found that a single mouthful of barium found its way promptly to the stomach if each act of swallowing were followed by a deep, well-sustained aspiration.

The stomach was of extreme "cascade" shape, presenting the typical illusion of having two cavities (Fig. 2). The globularly-distended posterior portion, or fundus, surmounted by an enormous gas-bubble, accommodated about 6 ounces of barium before its contents could spill over the anterior ridge into the relatively normal pars media. The latter, along with the pylorus, was displaced anteriorly and to the right by the distended colon. The duodenum exhibited normal contour and tonicity. (Unfortunately, film records were not obtained, because at that time there was no provision for the cost of x-ray investigation of relief patients).

Treatment was two-fold. (1) The patient was told again that he had no heart disease. It was explained, in simple terms, that he had acquired the habit of air-swallowing and that the stomach did not manufacture gas. The relationship of his illness to the loss of his job, as the precipitating psychological factor, was made clear. (2) Immediate return to normal diet was urged. After each mouthful swallowed he was told to take a slow, deep inspiration, since fluoroscopy had revealed the effect of diaphragmatic contraction on œsophageal emptying. Cathartics were prohibited, a saline enema to be taken if necessary. Rolling exercises and a rectal tube were recommended to aid the escape of colon gas. The response was gratifying. Next day he had much less belching and no substernal distress, but his wife complained that the ritual of taking a deep breath after each swallow prolonged the meal hour to almost two! In three days he was symptom-free, and fluoroscopy with barium showed the stomach now to be of ordinary J-shape, with normal clearance from the œsophagus, a small gastric air-bubble, and the left diaphragm at its usual level below the right.

DISCUSSION

In the deformity known as cascade stomach there is a pouching backwards and downwards of the fundus so that the stomach presents two cavities (Fig. 3)—an upper, spherical portion which must empty forward over a ridge or dam, to discharge its contents in cascade-fashion into a narrow, tubular portion representing the pars media. This latter is directed anteriorly, downwards and usually to the right, and exhibits normal peristaltic activity. The dilated fundus, however, seems able to empty itself in extreme cases only when the subject lies prone or on his left side. A large gastric gas-bubble, elevation of the left crus of the diaphragm, and gaseous distension of the splenic flexure of the colon are frequent concomitants. The symptoms are not specific—gas-belching, flatulence, upper abdominal distension, and fullness after meals. A bulging of the left thorax has been described.

Although cascade stomach was first explained on the basis of adhesions due to gastric ulcer, etc., the majority of the cases that have been examined at operation or autopsy have shown anatomically normal stomachs. Moreover, the condition is often observed to be transitory, and minor degrees of fundus-pocketing, or pouching, as it is also called, are seen in the course of x-ray examinations which yield no other abnormal findings. Barclay[1] suggests that with gaseous distension of the thin-walled fundus the oblique muscle fibres may remain in normal or increased postural tonus, thus creating the anterior wall of the pouch. Others, however, have felt that muscle spasm was excluded by the inefficacy of belladonna or papaverine. The literature has been reviewed recently by Upham,[2] who reports a case in which operation disclosed abnormal mobility of the fundus due to absence of the gastro-phrenic ligament. This lack of support he conceives to permit a posterior prolapse of the fundus, with rotation about the splenic artery and tail of the pancreas, as air-swallowing increases the intra-gastric pressure. Brohée[3] doubts the existence of an abnormally mobile fundus, but regards gaseous distension of the colon as of prime importance. This would seem to be so in the present case, with the additional supposition that distension of the gastric fundus is an equally important factor.

In reconstructing the sequence of events, the patient's loss of his job provides the psychological basis for the development of the neurosis. Why the physical manifestations were not tremor, insomnia, headache, palpitation or precordial pain, but were in this case gastro-intestinal, has not been determined. The onset of symptoms with belching, suggests that air-swallowing and air-sucking were among the first deviations from normal function. Aided by constipation, large amounts of air collected in the uppermost portions of the intestinal tract, namely, the fundus of the stomach and the splenic flexure of the colon. The buoyancy of the gas-distended limbs of the splenic flexure, lying as they do in the self-contained, semi-fluid system of the abdominal cavity, must exert considerable upward force. Directed

against the mid-portion of the posterior wall of the stomach, at a point below the gas-distended fundus, this would seem to be adequate to produce the cascade deformity. Whether one must postulate, with Upham, an abnormal mobility of the fundus, is a matter to be settled by operation and autopsy in the future. In any case, with further accumulation of air in stomach and colon the left half of the diaphragm was pushed higher and higher, becoming immobile during respiration of ordinary depth. It has been observed before that this may result in impeding upward belching from the stomach, either because diaphragmatic elevation of itself tends to close off the œsophagus at the hiatus, or because torsion at, or tension on, the cardia obstructs its orifice (Zollscher[4]). In the present case there was, in addition, serious interference with œsophageal emptying. Barium was retained to a height of 3 or 4 inches above the cardia for as long as five minutes, during which time the walls of the œsophagus became increasingly distended by swallowed air. Eventually the patient was induced by distress to gasp for breath, and with each forceful inspiration, as the diaphragm descended, the œsophagus discharged its contents. It was then found that if each act of swallowing was followed studiously by a deep inspiration, the material passed directly to the stomach, unaccompanied by excess air. With the cessation of air-swallowing, and the elimination of excess gas from both colon and stomach, the latter returned to its normal J-shape within three days.

It is concluded that the cascade deformity of the stomach in this case depended upon air-swallowing, a dilated fundus, and the distorting effect of a grossly distended left colon. The œsophageal disturbance was related to the resultant extreme elevation of the diaphragm. Treatment was aided by the elucidation, through fluoroscopy, of the mechanics involved.

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RADIOLOGICAL FINDINGS IN A CASE OF ACUTE PANCREATIC NECROSIS

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IN discussing this, the most alarming of the accidents met with in abdominal surgery, two points stand prominently forward as regards diagnosis and treatment. One is that he who first sees the patient immediately after the invasion of the pancreas has begun will probably receive an impression of the urgency of the condition which is not conveyed to those who may come in consultation later; the other is that the indications for the surgical treatment of acute pancreatic necrosis, in some cases, at least, will never be so apparent after a few hours have passed as they will have been in the first hours of one's observation. It is a matter of common knowledge that in a case of acute pancreatic necrosis we may find the patient passing into a period of relative comfort, once he gets beyond the period of the acute onset. In no other way can one explain our willingness at times to defer doing an exploratory operation, if at least one's instinct to cut down upon and drain the pancreas is opposed to the view still held by many physicians and surgeons that the operative treatment of acute pancreatic necrosis is without result.

As to the actual cause of an acute pancreatic necrosis there is still much debate. Self-digestion of the pancreas, due to activation of the trypsinogen it secretes, is the favoured idea, but one remembers that this ferment is only turned into active trypsin after the trypsinogen has passed from the gland into the duodenum, and come into contact with the enterokinase of the succus entericus, so that it is difficult to understand how the trypsinogen can become activated in the pancreas itself.[1] The generally accepted teaching that obstruction at the orifice of the common bile duct allows bile to be thrown up

into the pancreatic duct is again not to be too readily accepted, because in cases of pancreatic necrosis it is rare to find stone in the common duct and diverticulum of Vater, and it seems, according to some observers,[2] that the anatomical arrangements of the biliary and pancreatic ducts is seldom such as to allow regurgitation of the bile into the pancreas. Infection, as from disease of the gall bladder,[3] from a peptic ulcer, or through the blood stream and lymphatics, remains still as one of the most probable causes of acute pancreatitis,[4] and in the case here described the symptoms developed very shortly after an operation for the removal of a sequestrum which had formed on the right zygoma subsequently to an old wound.

How this infection may act is as yet obscure. Since, however, it seems to be accepted that trauma of the pancreas may precipitate a necrotic pancreatitis by bringing about the death of the traumatized tissue, it is possible that minute embolic accidents work in a similar way, and that the first result of a small infectious embolus is to produce a small area of simple necrosis from which may be derived the ferments necessary to convert trypsinogen into trypsin. That infection plays a distinct part in setting up an acute pancreatic necrosis seems further to be suggested by experiments which indicate that the injection of sterile bile into the pancreatic duct has but little tendency to create disturbance, whereas the injection of bile to which infecting organisms have been added seems fairly frequently to be followed by the symptoms of acute pancreatic necrosis.[2] It is possible that bacteria themselves initiate the transformation of trypsinogen into trypsin, bacteria transported at times from a remote focus.[5] In many cases, however, the cause of the acute lesion in the pancreas remains undetermined, as in the case hereunder detailed.

CASE REPORT

G., male, aged 52, was admitted to Christie Street Hospital on February 12, 1935, for a plastic operation upon the malar bone and infraorbital ridge on the right. Part of the malar bone had become detached and was lying free as a sequestrum. A plastic operation was done by Dr. Risdon under a general anæsthetic on February 16th. Ten hours later the patient was complaining of severe epigastric pain. He said also that he had headache and pain in the left upper lumbar region. He vomited actively and it was evident that he was suffering severely. The most careful examination at this time failed to note any rigidity or splinting of the abdomen. There was no limitation of the respiratory movements. His pulse was noted as 72 and his temperature as 99° F. There was no diarrhœa. The patient had given no history suggestive of antecedent biliary tract disease, ulcer, or appendicitis, and with negative findings on examination it was decided that such conditions as ileus, intestinal obstruction, embolism or thrombosis of mesenteric vessels did not come into the picture. He rested quietly for some hours after an injection of morphine and atropine. A slight degree of tenderness on deep palpation in the epigastrium persisted after the first symptoms. Pancreatic necrosis was suspected. The base of the left lung was collapsed. By next morning the patient looked very ill. He still had severe epigastric pain, was nauseated and vomited. Definite extreme tenderness in the epigastrium was now noted just below the xiphoid. For some hours on this second day there was slight splinting of the muscles in the upper part of the abdomen. The lower part was noted as soft and non-resistant, with no shifting dullness. The temperature this day was 98° F. The pulse rate, however, had risen to 100, and a blood count showed a very striking increase of the white blood cells (24,000), with 90 per cent of polymorphonuclear cells. Through the day the slight signs of rigidity disappeared and the tenderness in the epigastrium became less marked. Some slight resistance persisted, however, in this area and extended somewhat to the left. The nausea and vomiting, which had been most persistent, became less troublesome, and for a matter of six days hereafter a general improvement seemed to be in evidence, an improvement, however, which was somewhat belied by the fact that there still remained a sense of resistance in the left upper quadrant and midline of the abdomen, a slight tenderness on pressure, a slight degree of pyrexia (99 to 100° F.) a pulse rate ranging from 90 to 116, and a persistently elevated white blood count. No fullness or bulging had appeared in the left lumbar region.

During this period of questionable improvement the hope was entertained that a pancreatic necrosis, if existing, was beginning to resolve. It was considered that more active signs of peritoneal involvement would be showing themselves if a perforation of an ulcer into the lesser sac had taken place, while it did not seem probable that we had to do with some late results associated with gall-bladder disease, ileus, intestinal obstruction, or, perhaps, mesenteric thrombosis. Under these circumstances more detailed examination of the stomach and intestine was considered permissible.

A screen examination showed the left dome of the diaphragm to be relatively immobile and distinctly higher than the right. All suggestions of a post-operative collapse of the left lung seemed to have disappeared. There was no suggestion of fluid immediately underlying the diaphragm.

Since vomiting and abdominal distress had seemed to be subsiding, a barium meal was given and the following remarkable findings were obtained.

1. All barium administered remained in the cardiac end of the stomach, in all positions, including even the semi-erect. After five hours the barium administered was found remaining in the same position and showed no tendency to pass into the lower half of the stomach. Twenty-four hours later a screen examination showed the barium still in the same position and, further, that none of it had been passed on. There was no outlining of the lower part of the stomach or duodenum. We felt that the meaning of this retention of the barium was that fluid had accumulated in the lesser sac and by pressing forward was producing an evident obstruction. Following the giving of the barium there was some return of the vomiting, and it seemed somewhat difficult to explain a vomitus that was distinctly bile-stained if the obstruction was sufficient to prevent the descent of the barium. During all the examinations at this time only the slightest sign of peristalsis could be noted in the upper part of the stomach, while, as was natural, with no barium deposited below, little or nothing could

be made out as to the muscular movements towards the pyloric end of the stomach.

2. Careful manipulation (in the erect position) of the stomach over the pars media and pyloric portion seemed to produce a fluid wave in some well-filled pocket. The fluid producing this wave could not be made to mix with the barium in the cardiac end of the stomach and for this reason the possibility of this collection being outside the stomach was suggested, with the further suggestion that the fluid was contained in the lesser sac. By passing a duodenal tube, 700 c.c. of bile-stained fluid was removed, but no barium in visible amounts was returned. It was suggested that the openings of the tube had reached below the barium accumulation and were bringing up fluid which might have regurgitated from the duodenum. A screen examination following this washing seemed to show that the fluid wave had disappeared, but one noted the same large bulk of barium still remaining in the cardiac end of the stomach. A duodenal tube introduced again remained this time hidden in the barium, and one and a half hours were required to wash out the barium still remaining 24 hours after its administration. At this time a tube could not be induced to advance further into the stomach in any position in which the patient could be placed.

3. Seventy-two hours after this investigation had been begun a loaded duodenal tube was passed with the patient in a semi-upright position and went down into the pars media and pyloric antrum. Eight hundred c.c. of bile-stained fluid were removed. Two glasses of barium were swallowed beside the tube, but the fluid remained fixed in the cardiac end of the stomach. With the opening of the tube fixed in the pars media, barium introduced through the tube regurgitated at once into the cardiac end of the stomach. The tube was then passed into the duodenum. Barium introduced into the tube passed on now into the small intestine. The tube was left *in situ*. With the passage and manipulation of the tube some little barium was now deposited in the lower part of the stomach. A very pronounced dilatation of the organ was apparent, but peristaltic waves were strikingly absent.

The net result of these examinations, which luckily gave no distress to the patient, was that one could determine a marked degree of obstruction to the passage of barium involving the lower two-thirds of the stomach. That this obstruction was not absolute seemed evident from the fact that bile-stained fluid regurgitated, and that the duodenal tube could be made to pass through into the duodenum. A fluid wave suggestively due to accumulation of fluid in some sac adjacent to the stomach was noted, and, finally, when some barium was diffused over the lower part of the stomach by the manipulation of the duodenal tube it could be seen that peristalsis was completely lacking.

From the patient's history and from these interesting x-ray findings a diversity of opinions resulted. Our own impression was still that we were dealing with a pancreatic necrosis which had been followed by perforation into the lesser sac and by an accumulation of fluid which by its pressure and by paralyzing the muscles of the lower part of the stomach was preventing the barium or stomach contents from descending. We found it difficult to explain the ease with which intestinal contents were regurgitated. We could only assume that the musculature of the duodenum had retained its power.

To more than one experienced radiologist the plates presented great difficulties. It seemed to them that no collection of fluid existed in the lesser sac or under the left dome of the diaphragm. An acute inflammatory condition subsequent to peptic ulcer seemed best to explain the pictures as put before them, in spite of the fact that there had been nothing in the patient's history to suggest the existence of an old or acute ulcer.

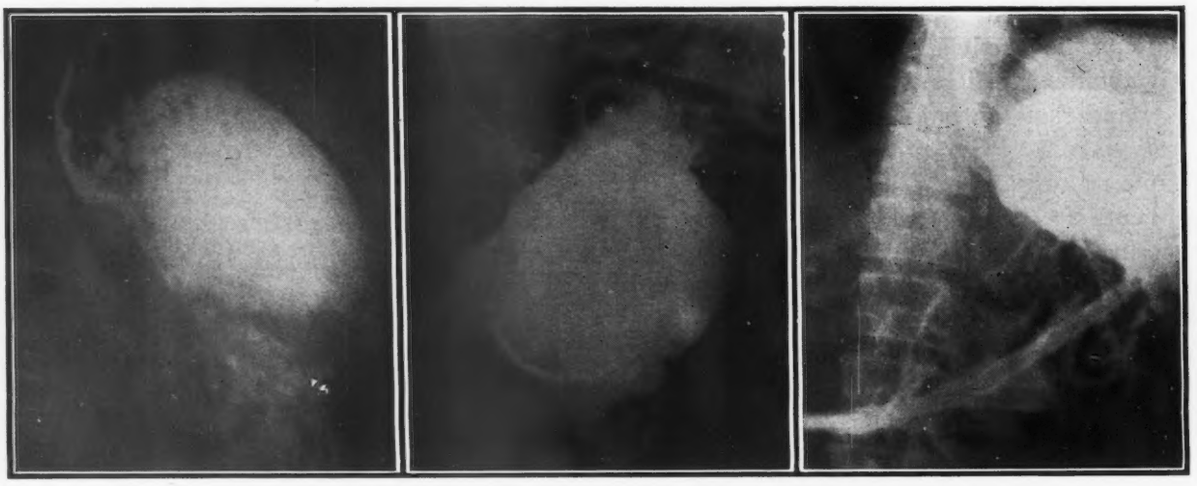

Fig. 1 Fig. 2 Fig. 3

Fig. 1.—Retention of the barium in the cardiac end of the stomach after 72 hours. **Fig. 2.**—Duodenal tube *in situ*, with barium still retained in the upper part of the stomach, though slightly displaced by an air bubble. Some flakes of barium are shown as if pushed down by the tube. Distinct wave of peristalsis along the greater curvature. **Fig. 3.**—Faint outlining of the pars media and pyloric end of the stomach. Free bismuth pushed down by the duodenal tube. No suggestion of peristalsis in evidence.

The opinions expressed by various medical consultants indicated that to them a pancreatic necrosis with lesser sac accumulation or an œdema of the stomach walls subsequent to ulcer were the conditions most to be considered. The surgical consultants veered strongly to the idea of the existence of an inflammatory œdema complicating a gastric or duodenal ulcer. The possibility of the development of a duodenal ileus was also considered. What was most apparent however in the expressions of all consultants was the fact that the pictures presented to them represented something of a most unusual nature.

During the examinations just detailed there was no appreciable change in the patient's general condition. That he was not making satisfactory progress was suggested by the persistence of a low-grade fever and a slowly rising pulse rate. Little or nothing could be felt in the abdomen; there was no prominence in the left lumbar region, a finding which has been at times reported in association with distension of the lesser sac. The

patient complained of no pain; there was still occasional vomiting. A slight degree of tenderness to deep pressure remained as a suspicious finding. Large quantities of bile-stained fluid drained away from the duodenal tube which was now left in place. The white blood count was now a little lower (11,000). Attempts at feeding produced vomiting. The various surgical consultants seemed unwilling to consider operative procedures. A note on March 6, 1935, says, however, that the barium meal then filled the pars media of the stomach and that some had even passed into the duodenum. Some slight peristaltic movements were noted along the greater curvature of the stomach and some barium could be seen passing on.

For the next four days a general improvement seemed to be in evidence, a normal temperature was reached, and small feedings and fluids were retained without vomiting. This improvement continued for about three days, when vomiting began again. On March 13th the note says, "still no appreciable change in general condition or in the abdominal findings". A surgical consultant considered any question of operation inadvisable, but two days later as a result of the patient having had a severe chill with drainage of fresh blood through the duodenal tube it was decided that a transfusion should be done and that an exploratory operation should be undertaken. Further suggestions of some serious progressive condition was evidenced by a rising pulse rate and a temperature which shot up to 104° F. At the exploratory laparotomy, made by Dr. Shenstone under general anæsthesia, on March 16th, one month after the operation upon the patient's face, and after the first onset of symptoms, much œdema of the gastro-colic and gastro-hepatic omenta was found. The stomach was adherent to the liver along the whole of its lesser curve. There was much bleeding, but no free blood or effusion in the general peritoneal cavity. The gall bladder and duodenum were involved in the inflammatory process, and there were small areas of fat necrosis in the omentum. A mass in the position of the lesser sac was thought to be an enlarged œdematous pancreas. The lesser sac was not explored. The abdomen was closed without drainage, and the diagnosis of acute necrotic pancreatitis seemed to be established. Following the operation the patient slowly sank; a continuous drainage of bloody fluid continued through the duodenal tube. Treatment, which consisted largely of intravenous injections of glucose and saline, produced no lasting result.

At the autopsy, of which the essential details only will be touched upon, the following most interesting findings stood forth. There was now some bloody fluid in the abdomen; the omentum was studded with areas of fat necrosis and was bound down to the liver, gall bladder and spleen. The foramen of Winslow was almost completely closed, save for a very small opening through which now bloody fluid flowed into the general peritoneal cavity. The gall bladder was distended, and seemed thickened, but contained no stones. The stomach was opened *in situ*. It contained about 200 c.c. of bloody bilious fluid. No ulcer was present. The posterior wall of the stomach was thin, and in several areas appeared as if it would perforate on the least manipulation. Tracing through the pylorus, one found the first part of the duodenum inflamed and congested, and, passing onwards, an ulceration was discovered, 1 cm. in diameter, which had destroyed the ampulla of Vater and had opened into the necrotic head of the pancreas. An easily-traced sinus led through the head of the pancreas into the lesser sac, and through this sinus fluid drained easily from the lesser sac into the duodenum. The head of the pancreas was completely necrotic and friable, the rest of the organ was swollen, hæmorrhagic, œdematous, with areas of necrosis here and there, and with numerous small specks of fat necrosis in and about the whole organ. The lesser sac of the peritoneum was distended and contained 500 c.c. of bloody fluid. The posterior wall of the stomach was roughened and necrotic. The head of the pancreas disintegrated completely during the examination, and one could find neither the opening of the pancreatic duct nor the lower end of the common bile duct. There were no stones in the bile ducts. Bile flowed into the duodenum and apparently found free access into the lesser sac through the ulcerated area at the site of the diverticulum of Vater and along the sinus passing through the head of the pancreas into the sac.

COMMENT

From these findings, one assumes that fluid and exudate from the lesser sac would drain into the duodenum and might regurgitate into the stomach. From the amount of fluid that might accumulate in the lesser sac there would doubtless be active pressure on the posterior wall of the stomach and from the thinned necrotic appearance of the stomach's posterior wall one assumes that peristalsis would be markedly inhibited.

As has been often noted in connection with pseudo-cysts of the pancreas the lesser sac of the peritoneum may fill and empty from time to time. With free entry of air from the duodenum, the occasional finding of a fluid wave, as was noted during the examination in our case, might be expected. A pressure sufficient to force the duodenal contents past the obstructing area in the lower part of the stomach might be generated by accumulation in the lesser sac, retention of duodenal muscle tone, plus the action of the abdominal muscles. In this way the vomiting of the duodenal contents might be explained.

Further examinations revealed the existence of diffuse necrosis of the pancreatic tissue with hæmorrhagic and inflammatory reaction, while the section of the stomach wall showed extensive necrosis and inflammation with wasting and destruction of all tissue elements.

CONCLUSIONS

1. A case of pancreatic necrosis is reported, in the course of which it was possible to carry out radiological examinations.

2. These radiological findings seem to be of an unusual nature, and do not seem to be described in radiological textbooks.[6]

3. The obstruction noted to the passage of barium, the failure of the lower part of the stomach to fill, and the absence of peristaltic waves in this part may suggest pressure from accumulation of fluid in the lesser sac and an inflammatory reaction in the stomach wall.

4. Pictures such as are here represented, obtained in a patient with a history of acute upper abdominal pain, should suggest occurrence of complications following pancreatic necrosis, such as distension of the lesser sac and involvement of the stomach wall in the necrotic process.

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A STUDY OF INTRACRANIAL HÆMORRHAGE IN INFANCY*

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WHETHER we like it or not, the biophysicist and biochemist have been gradually replacing the clinicians' art in diagnosis, and more particularly is this the case in large teaching centres. But in reviewing some of the literature on, and in analyzing a series of cases of, intracranial hæmorrhage in the new-born, I have been forced to conclude that as yet in the diagnosis of this condition the correct appraisal of prenatal conditions and the signs and symptoms presented post-natally are as important as is a report on the spinal fluid, if not more so. I do not wish to belittle the value of the findings of diagnostic lumbar tap, though it seems to be of uncertain import at times, as we know that intracranial hæmorrhage may be present with a negative spinal fluid and the presence of xanthochromia or blood in spinal fluid may not always indicate the condition under discussion. While it follows that the conclusion arrived at from the summation of the clinical picture and the report on the spinal fluid will be more nearly correct than a conclusion from either separately, I wish to stress the great importance of close and careful observation, particularly in those first few days or hours when treatment instituted early may do so much to cut down the dreadful tragedies of later life.

The premature infants.—The 34 infants in this class comprised 20 males and 14 females. Their average age was 7½ months, their average weight less than 4½ pounds. The type of labour was ascertained to have been rapid or precipitate in 14, difficult and prolonged in 14, and uncertain in the remaining 6 cases. Pituitrin was used in 10, and instruments in 13, both having been used together in 17 instances. There was one from each of three sets of twins, two breeches, and one Cæsarean case.

Thirteen were diagnosed presumptively, when they were presented about the age of eighteen months, as mental deficients, their record of delivery and neo-natal progress having been confirmatory. Eighteen were diagnosed by their signs and symptoms together with the results of spinal tap, and three were diagnosed at autopsy, there having been nothing clinically to suggest the condition prior to death. Nearly one-half the cases were therefore unrecognized in their early days. It is not always an easy matter to diagnose intracranial hæmorrhage in premature babies. As a rule, the smaller the infant, the less is the likelihood of diagnosis. Their smallness tends to make one hesitant about examinations and the handling required in lumbar and cisternal taps. The ordinary signs and symptoms present in the full-term infant are masked in the premature by listlessness, and persistent or recurring cyanosis, which tend to keep one's mind on systems other than the central nervous. The experienced handling of a small needle and the judicious use of a little blood, instead of the giving of tanks of oxygen would, we must agree, make our later problem a much easier one.

Regarding treatment, it was decided to categorize both premature infants and those at full term under the following headings: "A" untreated; "B" treated late and inadequately, where for example one or two taps were done late and blood either not given or given too late; "C" treated early and adequately where diagnostic lumbar or cisternal taps were done at the first warning and repeated until the fluid became clear, and where blood was given intravenously early and repeated if necessary. The results were classified; "A" died; "B"

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mentally deficient; "C" partially recovered; "D" completely recovered.

Seventeen of the 34 cases were untreated, and these included 13 mental defectives; 10 were treated late and inadequately; and 7 early and adequately. Six, in this latter class, made a complete recovery, 13 turned out to be deficient, and 15 died. Ten went to autopsy, 2 of whom showed a tentorial tear, 3 showed generalized bleeding intracranially with varying degrees of hæmorrhage throughout the viscera, and 5 revealed large clots over both hemispheres. With such a high mortality and, what is worse, such a high permanent morbidity, it must be admitted that these figures do not present a very brilliant result. Therefore it has been decided to perform diagnostic lumbar or cisternal taps in all premature infants as early as possible, regardless of the type of labour and clinical picture, and to establish early drainage and to give blood intravenously.

The full term infants.—There were 58 infants, born at term, diagnosed as having had intracranial hæmorrhage, 23 females and 35 males. Their average weight was slightly over 7 pounds, 9 ounces. Thirty-six were from primiparæ, 22 from multiparæ. The pre-natal condition of the mother in 10 cases was poor, in 10 fair, and in 38 good. Her average age was slightly under 30 years. There were 11 easy, 14 moderate, and 31 severe labours. Pituitrin was used in 18, and forceps in 21 instances. There were four breeches and one from each of two sets of twins. The foregoing facts support the greater importance of trauma as an etiological factor in intracranial hæmorrhage, though no attempt has been made in this presentation to uphold the argument as to whether trauma or hæmorrhagic disease may be the more important cause. Case 89 is here presented as a rather interesting case due to trauma.

CASE 89

Prenatal condition poor—one other child still-born and one other lived 18 hours. Delivery was non-instrumental, and labour long. The child was first seen at 10th day with history of fever and convulsions. Examination revealed upper respiratory infection. Convulsions continued. Signs of intracranial hæmorrhage were masked at first, but diagnosed a few days later by cisternal puncture.

Autopsy showed a tentorial tear with large clots infratentorially.

Sixteen of these infants were born in the months of June, July, August, September and November, 42 in the remaining months, when ultra-violet light is less intense. I have always had the feeling that hæmorrhagic disease was more frequent and severe in winter than in the summer months. Can low blood-calcium states, on the part of the mother as well as the new-born, have anything to do with this tendency to bleed? The case following is a rather typical example of hæmorrhagic disease of the new-born, as being causative of intracranial hæmorrhage.

CASE 84

Prenatal condition good. Full term and a non-instrumental labour which was easy. Weight 9 lbs. Cry, colour, actions, normal. During the first few hours there were cyanosis, twitchings, shrill cry; a slow pulse at the sixth hour. A cisternal tap revealed bloody spinal fluid. Immediate transfusion and continuous drainage were carried out. The child died at the eighteenth hour.

Autopsy.—Hæmorrhages into ventricles, subarachnoid space, intestines and viscera. No evidence of injury.

The family history in 7 instances was of interest. Three families gave a history of having had one or more still-births, one family of having had two children live only a few hours, and three families of having had one child turn out mentally defective. Two of these latter families are of special interest. Patient 6 of the series, now twelve years old, a case of Little's disease, had a brother born four years ago, and, being on the lookout for trouble, this brother was treated early and intensively with several transfusions and repeated lumbar and cisternal punctures. Despite this, however, at his present age of four he displays minor mental and physical stigmata. In the second family, case 22, eight years ago, was diagnosed as having intracranial hæmorrhage and treated successfully. This history was overlooked, and when his brother came along two years later (case 30), it was only when he developed rapid, shallow breathing, twitchings, spells of cyanosis, and tarry stools, that the earlier case was recalled. These points should suggest the importance from the point of view of predisposition to intracranial hæmorrhage of a family history of mental deficiency, still-births, and other proved cases of intracranial hæmorrhage or of hæmorrhagic disease, particularly with reference to preventive treatment.

The diagnosis was based on the clinical picture, corroborated by cisternal or lumbar punctures. As may be inferred, a correct appraisal

of the signs and symptoms is most important. A comparison of the cry, colour, action, breathing, sucking, etc., of these infants, except in a few instances where is was impossible to obtain information, was as follows: of the fifty-eight, 50 sucked poorly; 32 were lethargic compared with 18 who were restless; 38 were cyanosed, 10 pale, and 10 had blue spells; the cry in 30 was weak, in 15 shrill, in 4 whiney, and in 4 strong; breathing was rapid and shallow in 29, difficult in 20, and normal in 4; muscular twitchings were present in 36; convulsions, in varying frequency, were present in 22; 6 were limp, and many were noted to have had rolling eyes; 8 had dysphagia, and the early efforts on the part of the nurses were misdirected at clearing mucus from throats as a result. Case 86 was of interest, as demonstrating dysphagia.

CASE 86

The prenatal condition was good. A full-term child. Moderately severe labour with low forceps. Weight of child 7½ lbs. Mucus in the throat was troublesome from the first and suction was used frequently. On the 2nd day cyanosis was observed on attempting to swallow water or food. On the 3rd day the cyanosis continued, and the child would go into apnœa when feeding, stiffen, and after a few minutes gasp for air. Thymus disturbance and tracheo-bronchial fistula were eliminated. The child was treated for tetany with no improvement. On the 4th day, cisternal puncture. The fluid was xanthochromic and under pressure. An excellent response was made to repeated lumbar and cisternal taps. The decision was arrived at to perform immediate spinal tapping in all abnormal new births.

The fontanelle was tense in 17 and bulging in 2 cases. Overriding was present in 5 cases. The pulse was slow in 33 and rapid in 15 instances; local paralysis was present in 8 cases; there was evidence of bleeding into the skin, intestinal tract, etc., in 20 instances. An important sign was the early appearance of petechiæ on the palate, hands and wrists. Vomiting, though frequent, was not considered an important symptom, as so many new-borns vomit from various causes. Fever, though frequently present, was considered to be due to dehydration as a result of refusal of water and food, and not as directly symptomatic. One of the mentally deficients was probably so because of intense treatment to correct marked dehydration rather than drainage of spinal fluid.

With the exception of 8 cases all were investigated by lumbar or cisternal tap by myself or a well-trained assistant. I consider either procedure to be difficult and failures are not uncommon. The cisternal method is now preferred, but the one has complemented the other. The usual 21 or 20-gauge needle with short bevel was the one of choice, though in small premature infants a finer needle has been found more satisfactory. In some instances a Keidel vacuum tube has obtained fluid when other methods have failed. This latter is of special advantage when attempting to explore or drain a cortical clot through the fontanelle. Case 87 illustrates this procedure.

CASE 87

Prenatal condition good. A full-term child. Labour was precipitate; weight 6¾ lbs. The cry was shrill from the first. Slight cyanosis at times. On the 2nd day the heart rate was 90 to 100. Cisternal tap revealed xanthochromia with microscopic blood. On the 3rd day paralysis of left arm and paresis of left leg were noted; left facial muscles involved. Transfusion and repeated spinal taps were done, 18 in all. On the 11th day the paralysis appeared to be improving very slowly. On advice of Dr. Cone, of Montreal, a needle was inserted at the angle of the right fontanelle and 3 c.c. of broken-down blood aspirated. The paralysis disappeared rapidly. At 6 months the child was normal.

Very few ventricular taps were attempted. In the presence of the typical clinical picture xanthochromia alone was presumed to be due to intracranial hæmorrhage. Blood, unless it showed general crenation of red blood cells, old cells, and xanthochromia, was not accepted as confirmatory evidence. Thirty-nine cases revealed blood and xanthochromia, 10, xanthochromia alone, and one, clear fluid. The pressure was estimated to have been increased in 22 instances, but manometer readings, except in a few cases, were not done. Bleeding and clotting times were not carried out as a routine.

TREATMENT AND RESULTS

In 50 cases 246 lumbar punctures and 81 cisternal punctures were done, the average having been over 6. One patient had as many as 20 lumbar and cisternal punctures altogether. Continuous drainage was carried out in four instances. There may be considerable to say for and against this procedure but it has, at least, a considerable diagnostic value. In the following case this method was employed.

CASE 72

Prenatal condition good. A full-term child. Labour was easy, with low forceps. Weight of child, 7½ lbs. The condition was normal at birth; at eighteen hours intracranial hæmorrhage was diagnosed — hæmorrhages into skin and hæmatemesis. Immediate transfusion and repeated taps were carried out without improvement. A second transfusion was done and continuous spinal drainage was carried out. One hundred and forty c.c. of bloody spinal fluid drained off in four days without

improvement. The fontanelle was bulging. Old blood was obtained by the Keidel tube from both cortices through fontanelle. A third transfusion and an operation were done on the 5th day, to remove two large clots over each cortex. Recovery was immediate and good. At 2 years the child can talk and walk.

Blood was given intramuscularly in 15, intraperitoneally in 3, and intravenously in 48 cases, and in a few instances these methods were complementary. Intramuscular blood has a valuable place as a preventive measure, but it was never felt that, once intracranial hæmorrhage was diagnosed, the giving of intramuscular blood was adequate. Some infants were given as many as three transfusions. The conviction that the intravenous method was the best, despite some opinions to the contrary, was based on the marked improvement that followed its use. Small amounts, 5 c.c. per pound, were given and repeated in twenty-four hours if necessary. Punctures were carried out concurrently, and repeated as indicated by the severity of the case. The great advantage of the giving of blood and the performing of punctures at the first suspicion of bleeding must be conceded; blood not only to "buck up" a seriously shocked metabolism but to improve clotting power, and drainage to relieve pressure. Further treatment was secondary, consisting of absolute quiet, which meant no nursing at the breast, sedatives to control restlessness, twitchings and convulsions, and interstitial glucose to combat dehydration, etc.

The 58 cases were classified as to treatment and results under the same heading as the premature children. Eight of these 58 cases were untreated, 16 were treated late and inadequately, and 34 were treated early and adequately. Fifteen died, 12 turned out to be mentally deficient, 3 partially recovered, and 28 completely recovered. The decision as to the final result presented no difficulty, as all cases were followed for a year and a half, and in as much as they could not stand, walk, talk, etc., within the usual average period, they were placed in the completely recovered or mentally deficient classes, respectively. The three partial recoveries showed slight physical defects with minor mental impairment, but were not considered to belong to either category.

Of the 15 who died 8 went to autopsy, 3 of whom had hæmorrhagic disease, 3 tentorial tears, and 2 a torn falx with supracortical bleeding.

CONCLUSIONS

Intracranial hæmorrhage is difficult to diagnose in premature infants, and it would be advisable to do diagnostic spinal taps in all cases.

A family history of still-births, mental deficiency, or other cases of intracranial hæmorrhage or hæmorrhagic disease should cause one to take precautionary steps. The giving of blood intramuscularly is a very good preventive measure.

Forty-two cases of intracranial hæmorrhage of the new-born, in a series of 58, happened in the months when sunlight is of little value for metabolism. The question has been asked if this might be a factor in the production of easy bleeding.

Adequate treatment constitutes the early administration of intravenous blood in small amounts, and the early and repeated tapping of the spine at either the lumbar or cisternal site.

Continuous drainage may have a place in certain kinds of intracranial hæmorrhage. It is of value diagnostically.

There is the greatest need for the closest liaison between the obstetrician and pædiatrician in order that early and adequate treatment may be instituted.

"For we are made for cooperation, like feet, like hands, like eyelids, like the rows of the upper and lower teeth."

MARCUS AURELIUS

CALCIUM METABOLISM AND PREGNANCY.—According to R. Malméjac the fetus towards the end of pregnancy demands 1 to 6 grams of calcium daily, while an average maternal diet furnishes only 0.25 gram daily. The resulting negative calcium balance may lead to dental caries, osteoporosis, and defective lactation. Toxic conditions of pregnancy are to be attributed in part to deficient calcium intake, which in the early months may lead to nausea, vomiting, and anorexia, and in the later months to a syndrome of hepatic insufficiency akin to that of acute yellow atrophy. In pregnancy toxicoses, especially eclamptic, there is an impairment of hepato-parathyroid balance, which may be associated with hyperguanidinæmia, and which may be redressed by administration of large doses of calcium. Exhibition of parathyroid extract, vitamin D, and ultraviolet radiations are useful adjuvant corrective measures. The diet of pregnancy, besides being rich in calcium, or if necessary enriched by calcium medication, should contain milk, eggs, and butter; lactic fermentation favours assimilation of calcium.—*Bull. Soc. Obstét. Gynéc. Paris,* Oct., 1936, p. 575. Abs. in *Brit. M. J.*

THE USE OF APPLE POWDER IN THE TREATMENT OF DIARRHŒAL CONDITIONS AND ITS RATIONALE*

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IT is very interesting, if not significant, that of all fruits the apple is mentioned most frequently in folk-lore. There is an old Devonshire rhyme which says:

"Ait a happle avore gwain to bed,
An' you'll make the doctor beg his bread."

It is in the German folk customs that the original idea occurs for the use of the apple in specific ailments. In Westphalia apples were mixed with saffron to cure jaundice. In Pomerania, to eat apples on Easter morning is believed to insure against fever. In Hesse it is said that if an apple is eaten on New Year's Day it will produce an abscess. In Silesia and Thuringia an apple is scraped from the top to cure diarrhœa, and from the bottom to cure constipation. It was this custom that started Dr. August Heisler, about twenty-five years ago, to determine the value of apples in the treatment of infantile diarrhœas. The experiences of Heisler prompted Moro also to use the apple diet. Apparently, for many years, European physicians used the diet with great success. In 1930, Heisler reported his successes at the International Congress for Pædiatrics which was held in Stockholm.

During the War several interesting experiences occurred which later came to the attention of the medical profession and served to focus their interest again on the value of apples in the treatment of intestinal disorders. It was not long before these stories and the experiences of the continental physicians reached this country and excited, among pædiatricians in particular, great interest in the use of raw apple pulp. In January, 1933, Birnberg published the first report in this country on this type of therapy. Since then some seven or eight other reports have appeared in the American literature. A careful summary of all the case histories reported shows that 1,021 persons have been treated for various intestinal disorders with raw apple pulp. Of these, 1,005 showed complete recovery, while 16 failed to respond. This provides a mortality rate of slightly over 1.5 per cent. This is nothing short of marvelous when it is considered that many of the patients receiving this type of therapy were not placed on this treatment except as a last resort.

For the last three years this laboratory has been engaged in determining the various nutritive values of the apple.[1, 2] Altogether ten varieties have been studied. Their vitamin A content ranged from 12 to 36 units per ounce, and their vitamin C content from 1.5 to 7 units per ounce. It is now definitely established that vitamin A is necessary for the maintenance of a normal mucous membrane. For some reason, in the absence of this vitamin the mucus-secreting elements do not or cannot function, with the result that they are ultimately replaced by a different type of epithelium altogether. The value of mucin as a mechanical and chemical buffer against injurious agents needs only to be mentioned. Any agent, therefore, such as vitamin A, which serves in promoting normal mucus production is of great value in maintaining the resistance of the body against noxious chemical and mechanical influences.

In the course of our work it soon became apparent that we would have to prepare an apple product that would remain stable in its appearance and composition. As is well known, fresh apple pulp rapidly becomes discoloured. This so alters its æsthetic value that patients often refuse to take it. The oxidative and fermentative changes that occur soon produce such alterations that it is very difficult, if not impossible, to duplicate chemical determinations. We therefore prepared an apple powder and limited all future work to the use of this material. Our first powder was produced in January, 1934. In reviewing the German literature, we discovered that Wiskott, in July, 1931, had apple powder prepared. We did not succeed

* This paper was read at the combined meeting of the Pædiatrics Section of the Canadian Medical Association and the Pacific Northwest Pædiatric Society held in Victoria, B.C., June 24, 1936.

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in getting a sample of this until March, 1935. The German powder, "Aplona", contains the apple peel, while our product is made from the cored and peeled apple. Unless the residues of arsenic and lead are carefully removed, powder containing the apple peel should be subject to some suspicion.

In Europe the apple diet has been used successfully in the treatment of cœliac disease, cholera infantum, acute and chronic mucous colitis, summer and infectious diarrhœa, dysentery, acute and chronic dyspepsia, acute and infectious enteritis, paratyphoid and typhoid fever. In this country the diet has been employed chiefly in the treatment of various diarrhœas and dysenteries. Very little effort has been made by the medical profession to extend this type of treatment to adults afflicted with similar disturbances. Whenever it has been tried, however, a fair degree of success has been obtained.

The use of apple powder in place of the pulp has met with the same success. Barondes,[3] of San Francisco, has employed it successfully in 47 cases of young nurslings and small children, and in 52 adults. Various clinicians whom we have supplied with the powder uniformly report favourable results. Dr. L. Howard Smith,[4] of Portland, Ore., describes the following very interesting case.

> The patient was a premature infant 11 days old. Its birth weight was 4 pounds. Since breast milk was not available the child was given an evaporated milk formula, with Karo added. It lost weight and developed a severe diarrhœa, so that on the eleventh day of life it weighed only 3 pounds. At this time the diet was changed to ten ounces of skimmed milk plus ten ounces of 10 per cent apple powder water. Feedings were given by gavage every four hours. Although the baby appeared in too desperate a plight to survive it is interesting to note that after three feedings of the apple powder in the formula the stools became less frequent and were firmer in consistency. The next day the child had only one stool, which was normal in appearance.

Dr. C. Ulysses Moore,[5] also of Portland, Ore., has treated with success over a score of children suffering from diarrhœal conditions. While treating vomiting cases, he noted that those children who could not retain the apple pulp were able to keep down the powder. Many young patients on Dr. J. B. Bilderback's[6] service at the Doernbecker Hospital have responded most excellently to this type of treatment. The response of adults suffering with chronic ulcerative colitis and diarrhœal conditions secondary to gall-bladder disease seems to be most encouraging. Dr. Noble Wiley Jones,[7] of Portland, Ore., also reports good results in such cases with this type of therapy.

A number of reasons have been assigned for the benefits derived from the use of the apple diet. These factors may be divided into five groups: (1) acids; (2) sugars and starch; (3) cellulose and hemicelluloses; (4) vitamins, and (5) pectin. Most of those who have had experience with the apple diet are inclined to believe that each factor contributes to the success of the treatment, and are not willing to assign the value exclusively to one. A review of the above factors will show that their value has been recognized before in the use of sour milk or buttermilk, barley water, rice water, glucose, vitamins, etc. It is surprising that one food substance such as the apple should combine all these factors. It is true that the apple is not the only fruit that has been used with benefit in the treatment of these disorders. Kohlbrugge, for example, used lemon juice for its acid value in the treatment of summer diarrhœas. Schachter used pears successfully, and they are far from being an acid fruit. Another non-acid fruit, the banana, has been used for many years in the treatment of cœliac disease. Grodecki used tomato juice for thirty-five years with success. He attributed its value to its acid and vitamin content. Malyoth ascribed the chief value of the apple diet to its pectin content. Any benefit derived from pectin content of such fruits as the orange or lemon would be relatively small, since this material is resident chiefly in the peel, which is not eaten, while the pectin content of the apple lies in the edible part. From this it will be seen that the therapeutic value of any fruit other than the apple depends upon how many of the above-mentioned factors it possesses. The apple, since it includes all these factors, occupies first place as a therapeutic agent of this type. This may explain the unique and ubiquitous place this fruit occupies in folk-lore.

1. *Acids: tannic, malic, acetic, butyric and lactic acids.—Tannic acid,* because of its astringent effect, forms a protective coating on the mucous membrane. Any therapeutic advantage that tea may possess is due to its content of tannin. The tannin content of the apple is unaltered in the preparation of the powder. Its concentration, however, is increased, since it

requires 100 grams of raw apple to make 15 grams of powder. Tannic acid, however, cannot be the sole therapeutic agent since it has no effect on the severe colic, which is almost immediately relieved when apple pulp is administered. Tea is a good source of tannin, yet it possesses very little therapeutic value in the treatment of intestinal disorders, and it is a well-known fact that such commercial preparations as tannalbumin have very little value. Notwithstanding these objections, Heisler felt that tannin substances are of definite benefit.

Malic acid as well as the other organic acids is not appreciably altered in the preparation of the powder. There is some evidence in the literature to the effect that malic acid possesses a definite antiseptic value. It would seem, if this were true, that it would be due to the acid reaction it helps to maintain. This would be equally true for the other acids present in the apple. The pH of the apple is fairly constant with a range of 3.3 to 3.75.

In the intestine the anaerobic activity of bacteria on cellulose produces *butyric, acetic* and *lactic acids.* A bacterium has been isolated from normal human fæces which decomposes cellulose and shows the extraordinary property of being unable to utilize any other carbohydrate. The products of its action in pure culture are acetic acid, lactic acid, ethyl alcohol and some butyric acid. Lactic and formic acids are produced as the result of bacterial activity on hemicelluloses. It is assumed that the pentosans are hydrolyzed to pentoses, and these in turn are fermented to produce butyric, lactic and acetic acids. Considerable quantities of the lower fatty acids will thus be produced in the lower intestine. Of these butyric acid plays the most dominant rôle in bacteriostasis.

Bergeim[8] has shown that one of the most toxic substances for yeast and *B. coli* found in the large intestine is butyric acid. Bergeim, Hanszen and Arnold[9] showed that when eight bananas replaced an equivalent amount of carbohydrate in the daily intake of normal persons that there was a marked increase in the butyric acid content of the large intestinal contents. Correlated with this increase in butyric acid was a relative decrease in viable *B. coli.* The Gram-positive to Gram-negative ratio of direct smears from the washings showed a preponderance of Gram-positive forms. (Dysentery, typhoid, *B. coli* are Gram-negative, while cocci, acidophilus and clostridium groups are positive.) Bergeim[8] has shown that the concentration of butyric acid in the large intestine is perhaps the most important single factor in reducing the number of viable bacteria.

On the high banana diet amounts as high as 578 c.c. of 0.1 N butyric acid were found in the colon, and the average amount was 329 c.c. The action of butyric acid is amplified by the presence of lactic and acetic acid. When 70 g. of apple powder were ingested one hour before the introduction into the stomach of 520,000,000 *B. prodigiosus,* Bergeim and co-workers[9] were unable to recover from the colon any of these organisms eight hours later. When however 70 g. of banana powder preceded the ingestion of 335,000,000 bacteria, 0.044 per cent were recovered. Fresh banana and fresh apple both caused a marked reduction in the percentage of organisms recovered from the colon.

The beneficial effect of encouraging the growth in the intestine of lactic acid-producing organisms resides to a large extent in the ability of an acid medium to change the flora from the putrefactive to the fermentative type. This is of distinct advantage since the products of putrefaction are quite toxic, while those of fermentation are relatively harmless.

Usually there is a marked reduction in acid production in the stomach in diarrhœas and dysenteries. This is especially true if the disturbance is accompanied by an elevated temperature. The presence of organic acids in the stomach helps to maintain an acid reaction, thus promoting protein digestion and increasing the value of the "gastric barrier". Not only do food acids help in producing an optimum pH for peptic activity, but, by reducing the buffering effect of proteins, permit an optimum pH to be reached more quickly. Lowering the pH makes possible a greater ionization of calcium, which, in turn, exerts a beneficial effect by altering intestinal permeability and soothing the inflamed surfaces. Further, an acid reaction is essential for developing the full colloidal effect of pectin. In addition the organic acids have a certain caloric value and usually carry with them alkaline minerals which are of value in replacing those lost in the stools. The tart taste of the apple provides a pleasant way for the administration of acids.

Arnold[10] has shown that the self-disinfecting process in the upper part of the small intestine depends in some way on the presence of acid-buffered material. Normally, the acid reaction is maintained by the secretion of the stomach and by the choice of foods. In disease conditions, especially those characterized by an elevated temperature, the acid production of the stomach is reduced so that more emphasis must be placed upon the selection of foods. Hot environments, fevers, alkaline foods such as milk, and protein-rich foods such as meat, cut down the self-disinfecting mechanism by reducing the available acid.

While it is undoubtedly true that the acids of the apple have a decidedly beneficial effect, the therapeutic value of this food material is not totally dependent upon them. Kohlbrugge supports the idea that the chief value of the apple diet lies in its acid content, and administered lemon juice to adults suffering from diarrhœa with, he claims, good results. He states further that it is the acid that confers upon sour milk and buttermilk its therapeutic value. On the other hand, Kollman treated small infants with lemon juice with poor results. When he changed to apple improvement was immediate and striking. Freudenberg administered lactic acid, citric acid, phosphoric acid, and other acids and was not able to demonstrate any beneficial effect. Malyoth neutralized apple sauce and obtained results just as good as if he had used fresh apple. These experiments should not be considered as proving that acids in general have no value, but rather that the lower fatty acids such as butyric acid have a specific value and that the apple possesses more than one therapeutic agent.

2. *Sugar and starches.*—The available sugar content of the apple is a little over 12 per cent. It is composed of glucose, sucrose, fructose, and a small amount of starch. Aside from its caloric value, sugar increases the colloidal properties of pectin. Oxidation of this material in sufficient amount will prevent acidosis.

3. *Cellulose and hemicellulose.*—The cellulose content of the fresh apple is slightly over 1 per cent, while that of apple powder is a little over 8 per cent. One of the chief values of cellulose lies in its ability to provide bulk. Moro says, "The passage of the spongy, filling masses through the intestine frees it both by absorption and mechanical action of various injurious substances. In the treatment of infectious diseases of the small intestine detoxication is in this regard understood without further explanation. It is in this coarse method of cleansing that I see the principal therapeutic factor." Heisler advises leaving the skin on the apples, believing that it enhances their value. Moro attributes the benefit of the apple diet to the absorbent powers of the indigestible material, and to the fact that it causes an irrigation of the intestine with large stools. He feels that in this way injurious bacteria and the substances they produce are removed.

Kohlbrugge does not believe that the factor of bulk is very important. He states that the use of rice flour and arrow-root for their mechanical value is of little benefit. Malyoth, too, places very little importance on cellulose. He fed strained currants, which are rich in pectin and which are cellulose-free and obtained results which were in nowise impaired by the removal of cellulose. In this connection, he quotes the following interesting letter.

> "We had been in poor quarters. The food supply was low and the water so poor it had to be filtered of its mosquito larvæ before it could be used for drinking purposes. In time we were all suffering from diarrhœa, with numerous bloody stools. Soon a command was given to advance against the enemy. On the march we came upon a grove of currant bushes, all bearing a load of red berries. Regardless of commands or enemy bullets, we all threw ourselves under the branches and ate the fruit in great amounts. Immediately the intestinal pains were stopped, and in three days we were well."

There are present in plant materials at least two hemicelluloses[11] which have a nutritional value. One hemicellulose contains xylose in association with glucuronic acid, and the other contains arabinose in association with galacturonic acid. The more lignified the tissue, the greater the xylose and glucuronic acid content,—the less lignified, the more it contains of arabinose and galacturonic acid. The values of these uronic acids will be discussed in connection with pectin.

4. *Vitamins.* — Grodecki and Currado both mention the value of the vitamins contained in apples. For the most part these are limited to vitamins A, B and C. Work of the last few years has demonstrated conclusively the value of vitamin A in the maintenance of a normal

mucous membrane. Any condition where there is a large water exchange increases the need for water-soluble vitamins. Vitamin B is known to exert a marked influence on appetite and on gastro-intestinal tonus. Vitamin C, or, perhaps, as it should be called, cevitamic acid, is so constituted chemically that it undoubtedly plays a very powerful rôle in the detoxication mechanism. Evidence is accumulating that vitamin C is definitely protective against the effect of diphtheria toxin. Our own work shows that vitamin C when given to animals made toxic with menthol increases their ability to excrete this material in a conjugated form.

5. *Pectin.*—The chief value of the apple in the treatment of diarrhœas and dysenteries appears to reside in the pectin component. Pectins are hydrophilic colloids possessing high absorptive ability. It is partly due to this latter quality that pectins are able to fix bacilli and toxins and thus render them inert. By the same property it is able to take up the fluid secretions and thereby convert a fluid stool into a formed one. It is believed that the excessive peristaltic activity of the intestine during diarrhœa is due to the presence of irritants. Pectin removes the irritants and by providing bulk stimulates a normal physiologic peristalsis. It is to the pectin component of food materials more than to the cellulose or fibre that fæces owes its bulk. Pectin is also a good buffer, and through the medium of this activity is able to keep the fluctuations in the reaction of the intestinal contents within narrow limits. It also acts to some extent as a protective colloid. The pectin content of fresh apples averages about 5 per cent. The pectin content of apple powder is increased chiefly at the expense of cellulose. The following figures illustrate the greater amount of pectin in powder than in the fresh apple.

Variety of apple	*Fresh apple*	*Apple powder*
Jonathan	2.08	2.45
Winesap	3.12	5.33
Spitzenberg	4.27	6.07
Yellow Newton	5.55	10.99

Malyoth believes that the increased potency of the powder in the treatment of diarrhœas is due to the doubling of its pectin content. This same observation provides evidence against any great participation on the part of cellulose, since it is reduced in quantity, and on the part of tannin and organic acids, since they remain fairly constant.

When pectin is isolated from the apple and administered to patients suffering with diarrhœa the abnormal conditions disappear in the same length of time as if the original apple had been used. Increasing the dosage of pectin beyond that necessary provided no additional advantage. When luizyme, a ferment which destroys cellulose and pectin, is first added to pectin the resulting product is without effect. Commercial pectin is not as efficient, because of the evident loss of some of its characteristics. It absorbs less well, is a poorer buffer, and forms less bulk in the intestine.

The physico-chemical values of pectin may be summed up as follows. (1) It possesses great absorptive capacity for bacteria and toxins; (2) because of its colloidal nature it is capable of taking up large quantities of fluid; this provides bulk which helps to sweep out of the intestine harmful materials and to provide a normal stimulus for peristaltic activity; (3) by its buffer action it helps to maintain a constant reaction in the intestine; (4) it acts as a protective colloid to an inflamed and perhaps ulcerated intestinal mucosa.

The chemical characteristics of pectin are equally interesting. The molecule contains eight molecules of galacturonic acid. This acid is an isomer of glucuronic acid, which is known to form conjugation products with many toxic materials. That pectin is broken down in the digestive tract is indicated by the presence of methyl alcohol in the urine. The chief chemical value of pectin lies in the ability of galacturonic acid to form conjugation products with toxic materials. Until now the evidence that galacturonic acid could participate equally as well as glucuronic acid was not well established. It is this particular point that has been cleared up by the work in our laboratory. Thus it will be seen that there are present in foods such as the apple two uronic acids, both of which possess the remarkable ability of combining with toxic materials to render them inert.

SELECTION AND PREPARATION OF THE APPLE

For the most part raw apple pulp has been used, although Heisler states that cooking the fruit has no effect upon its therapeutic value. Unsweetened applesauce and whole baked apple were almost as effective as the raw pulp. Moro feels that when the cooked apple is used the beneficial action is retarded and the results are less encouraging than with the raw material. While Moro recommended ripe mellow apples, Heisler felt that ripe green apples which were quite sour were the ones to be chosen. There is very little evidence in the literature to indicate that there is any difference in potency that can be ascribed to differences in the variety of the apple. Most workers report that any variety is equally good, although there are a couple of statements to the effect that the Gravenstein is of little or no value. Perhaps the unfavourable results obtained by a few men have been due to the unfortunate selection of a variety that possessed very little potency. A table follows showing the uronic acid content calculated as glucuronic acid mg. in 10 g. of

fresh fruit. The uronic acid was determined by two methods. One was that described by Bang,[12] modified to use the Shaffer-Somogyi copper reagent. The second method was that described by Dickson *et al.*[13] The number of Sherman units per ounce of vitamins A and C is also included.

Variety	*Uronic Acid*	*Vitamin A*	*Vitamin C*
Gravenstein	73.1	14-17	3.5
Jonathan	85.1	24	1.5-2.0
Baldwin	90.8	15	2.5
Stayman	102.6	17	3.0
Arkansas Black	106.5	36	2.5
Delicious	118.0	24	2.5
Spitzenberg	121.4	35	7.0
Rome Beauty	133.3	12	2.5
Winesap	139.5	26-30	2.5
Yellow Newton	164.9	34	6.0
Tomato juice filtered	20.0		
Orange juice filtered	40.0		
Lemon juice filtered.	20.0		
Winesap juice	54.3		

In the preparation of the powder it is probable that a large portion, if not all, of vitamins A and C are destroyed. On the other hand, the pectin content is greatly increased. Outside of the loss of a major portion of vitamins A and C the balance of advantage lies largely in favour of the use of apple powder. The advantages may be summarized as follows. (1) the powder represents a product of high and uniform potency; (2) the æsthetic difficulties of taste and colour are overcome; (3) cooperation of the parents will be more readily attained. Wiskott says, "It is no easy problem to convince a mother that the very raw fruit which has often enough been the cause previously of diarrhœa among her children would also correct this very disorder"; (4) the bulk of the apple pulp makes dosage difficult; (5) the pulverized cellulose threads of the powder make a smoother stool; (6) the powder can be added easily to milk formulæ and will produce no curdling; (7) it represents a product of uniform price, available at all times of the year; (8) it keeps well, is light and compact; (9) it is not rejected when given to children who are vomiting.

In conclusion let it be said that apple powder represents a very potent preparation in the treatment of intestinal disorders. It has its greatest application in infantile conditions, but is finding a definite field of usefulness in the treatment of gastro-intestinal diseases in the adult. The value of apple powder is not resident in a single factor. Its usefulness is due to the summative effect of several potent elements. Its availability and ease of administration should recommend it for more general use.

The two following case histories are typical.

CASE 1

On October 23, 1935, a male child, aged two years, entered the Doernbecker Hospital. His temperature was 104° F., and the impression gained from the examination was that he was suffering from acute upper respiratory infection. The following day the patient passed seven stools in eight hours. The stools were loose, green and foul-smelling, and were mixed with blood and mucus. The temperature was 101° F. The patient complained of epigastric pain. Up to October 27th, the stools averaged seven per day. At this time, 10 per cent apple water was administered. In twenty-four hours, the stools had decreased to four per day, but they still contained some mucus and blood. The general condition however was much improved. By October 30th the stools were averaging three per day, were loose and yellow, and contained some blood and mucus. On November 5th the general condition was so improved that scraped beef, toast, broth, and eggs were given in addition to the apple water. On November 7th the diarrhœa had disappeared, although the temperature was still slightly elevated. A week later the patient was discharged. His stools numbered two a day and were usually soft, brown and large.

CASE 2

T.R., aged seven weeks, entered the Doernbecker Hospital on July 16, 1935. For two weeks prior to admission the child had suffered from severe diarrhœa, averaging ten to fifteen bowel movements per day. Upon admission, the patient was placed on a formula of evaporated milk and barley water. Two days later (July 18th) the patient had 32 bowel movements. The stools contained blood, were dark green in colour, and were not foul. On July 20th the formula was changed to skimmed milk, 15 ounces, 10 per cent apple water, 15 ounces. Four ounces were given every three hours. The child was continued on this diet for four days, at which time the stools averaged 2.5 per day, contained no mucus, and were soft and of a greenish or brownish colour.

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EXPERIENCE WITH FIFTY-SEVEN BRUCELLOSIS INFECTIONS IN SASKATCHEWAN*

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Saskatoon

BRUCELLOSIS is at present the term of choice for that specific group of infections classified as "undulant fever". It is a more inclusive term intended to incorporate the variations in both cause and course of the disease. The nomenclature evolved with accumulating knowledge has become cumbersome, confusing, and is not sufficiently comprehensive. The particular reasons for this report are twofold. Laboratory information exceeds clinical knowledge, consequently the problem is still that of sifting evidence in order to define the disease entity. It is entirely possible that geographical influences may be diversifying further the symptom pattern.

HISTORICAL

An epidemic form of fever in man had long existed in the Mediterranean area and was especially noticed in the island of Malta. It was first described by Marsten in 1861 and called "Mediterranean fever".[1] But it was not until 1886 that Sir David Bruce proved the etiological factor, which he called *Micrococcus melitensis.* In honour of Bruce other organisms belonging to this group have been given the generic name of Brucella. Twenty years later it was recognized that the goat was the host, and that in herds this organism was the cause of abortion. In the human being the disease was variously termed Mediterranean, Gibraltar, Malta, undulating and undulant fever. It was reported in America as early as 1897 and since epidemics were restricted to regions where goats were largely handled, it received the popular name of "goat fever".

Bang, a Danish veterinarian, succeeded in isolating the causative agent of abortion in cattle in 1897. Its pathogenicity for man was however not fully recognized until nearly twenty-five years later, although earlier, in 1911, Schroeder and Cotton reported finding the abortion bacilli of Bang in milk sold for consumption, and suggested this might result in human infection. In 1914 Traum isolated the causative agent of abortion in swine. The question was not raised for some time afterwards whether there was any relationship between these organisms either to one another or to man.

Heretofore, three organisms were known to effect separately abortion in goats, cattle and hogs. Only one of these, *Brucella melitensis,* the caprine or goat variety, was already proved a cause of fever in man. In 1918 Alice Evans, at Washington, demonstrated the intimate morphological, cultural and biochemical relationships of the goat and cattle (Bang's) organisms. In consequence of this investigation the Brucella-like organisms originating from goats, cattle and swine were designated as varieties of the same genus. Then, in 1924, Keifer in Baltimore reported a case of undulant fever in a patient who had had no contact with goats. The infection was the bovine type. Since then the porcine strain has also been recovered in human disease, and even known to be more virulent and more readily acquired from contaminated material, and not only by ingestion.

Briefly, the recognized principal species of the Brucella genus are three in number: (1) *Br. melitensis* (caprine, goat); (2) *Br. abortus* (Bang's, bovine); (3) *Br. suis* (porcine, swine). Confusion comes from variably calling the organisms a micrococcus, cocco-bacillus and bacillus. There is a great deal of similarity. Morphologically, dissimilarity appears greatest in actively growing young cultures of *Br. abortus,* which curiously enough develop a bacillary form. The host specificity is not absolute, and that is important. For example, the *Br. suis* may affect cattle and infect man. Also infection may come from sheep. All three varieties of Brucella have been isolated from cattle. The transmission from one human being to another by contact is a possibility, in view of Amos and Poston's finding Brucella in the stools of patients. Without further details in this connection it is noteworthy that all varieties of Brucella give an agglutination reaction against a common antigen.[2]

LABORATORY DIAGNOSIS

The onus of proving Brucella infection rests on laboratory findings, although some justify the claim that preponderant clinical signs are sufficient for diagnosis where the laboratory fails in confirmation. That is probably to some extent true, but the diagnosis nevertheless always remains unconvincing. A disturbing circumstance is the discrepancy in what workers and different laboratories choose to call a positive reaction. And then an indisputable laboratory report does not mean the solution of the diagnostic problem in a good many cases. This may not yet indicate what is primarily wrong with the patient, especially where there is other recognizable disease. There are several laboratory tests, but only those with which we have some experience will be mentioned.

* Read at the Annual Meeting of the Saskatchewan Medical Association, Saskatoon, September 24, 1936.

1. *Blood culture.*—Recovery of the organism from the blood or excretions is positive proof. Cultures however are frequently negative in acute cases (Evans), and the chances of obtaining a positive culture from chronic cases are not good. This test is generally impracticable because of the large percentage of failures; the time required may be from 10 to 30 days; it is costly, and there are other obstacles. The value of a negative report does not compare with that of a positive.

2. *Serum agglutination.*—This is the commonest method. A wide range of opinion exists as to the dilution which constitutes a positive result. A titre of 1 in 80 is most commonly accepted, but as low as 1 in 10 is still recognized. It is shown in severe cases proved by culture that agglutinins may be lacking in the serum. This is even more likely to occur in mild cases. The percentage of failures is quoted as high as 16.6 per cent.[3] While it is vexing not to find an accepted standard for a positive titre, a generous balance of about 85 per cent dependability is left, so that it is the preferred test. The strength of the dilution at which the reaction occurred should always be ascertained. A simple statement that the result is positive is of limited value. Angle mentions that 15 per cent of tularæmia patients show a cross-agglutination reaction with the abortus organism.[4] In this series difficulty was experienced in connection with a *B. paratyphosus B.* infection, *B. typhosus,* and in one case which ultimately yielded a positive hæmolytic streptococcus blood culture.

3. *The intradermal test.*—One-tenth of a c.c. of a killed Brucella culture, standardized and diluted according to the amount of concentrate, is injected intracutaneously. Positive skin tests are obtained where agglutination is at 1 in 20 or even negative in the ratio of approximately 20 to 6 in a group of studied controlled cases . . . Beattie and Rice.[5] It is a simple and attractive test as a routine procedure but dependable killed concentrates are required and sloughing is not uncommon.

4. *The rapid method.*—The patient's serum mixed with a killed dense culture concentrate of the organism on a glass slide gives a ready rapid reading. Again, the positive is significant and the negative is of doubtful value. Thirty-four rapid method tests yielded 28 positive and 6 negative reactions, but in all the agglutination tests proved positive. In other words about 17.5 per cent negative rapid method results still remain doubtful.

5. *White blood cells.*—Leucopenia and relative lymphocytosis are frequently mentioned. In two equally acute cases 25,000 white blood cells per c.mm. were present in one and 2,000 in the other. Actually, in this series the figures for leucocytosis and leucopenia were about equal.

ANALYSIS OF CASES

Late in 1929 we first realized the possibilities of brucellosis as a clinical phenomenon to which people in this farming country would be specially disposed. At that time the laboratory was depended upon entirely to establish the diagnosis. Only selected problem cases were ever considered. For various reasons, and without getting results for a long time, this search was abandoned. A little over two years later Case 1 formed the basis for renewed efforts. The triad of fever, rapid loss of weight, and sweating, however, still remained the inducement for either agglutination tests or blood cultures. Then over a long period of time only four bacteriological confirmations were obtained. With improved laboratory facilities opportunities were extended for investigation of the less classical types and the so-called ambulatory patient. For want of conviction, clinical suspects not confirmed by laboratory tests are not herein recorded. On the other hand, three well persons in one family, contacts, are included. One member of the same family had active infection. The three contacts showed just as strong *Br. abortus* agglutinations, but were without symptoms.

Fifty-seven patients were positive clinically and serologically. Since the search was extended 2,492 records of patients seen on account of ill-health from various causes were reviewed. On 512 patients blood agglutinations were made on suspicion or as a routine, which is nearly one out of every five patients. Positive tests were obtained in 57, that is, 11 per cent of the 512 blood tests were positive. What is more striking is that 2.3 per cent of all sick people (only one-fifth had blood submitted for the test) were infected with Brucellosis in some form.

At this point independent figures from the laboratory standpoint for approximately the

same area in Saskatchewan are of interest. J. S. Fulton[6] quotes in 5,023 tests for Wassermann reactions 81, or 1.61 per cent, as showing positive *Br. abortus* agglutinations, and in 461 blood sera requisitioned for *Br. abortus* 40, or 8.61 per cent, were positive. In comparison with an approach to the diagnosis from a more inclusive clinical aspect, this difference seems amply justified. (The standard used is positive 1/50 plus at least a trace 1/100). In the course of investigation difficulties were encountered in three instances already referred to in regard to early suspicious agglutination results, ultimately established separately as *B. paratyphosus B., B. typhosus,* and hæmolytic streptococcic septicæmia. These are not included, as are also seven others which will be mentioned only in passing as worthy of note because such experiences cause skepticism. Contradictory serological reports were obtained on the same patients at the same time. In all cases many of the blood specimens were divided and submitted to two and frequently three sources for our laboratory information. While these are excluded in this analysis, such failures are not entirely questionable accuracies as they may be factors due to delicate culture strains, of which 127 varieties were isolated in Iowa.

From our experience we hesitate to employ the terms, intermittent, ambulatory, malignant or subclinical types. It appears, quite simply, that brucellosis is either acute or chronic. In both instances for example patients have been seen when ambulatory.

Acute cases.—The percentage of acute forms in this group was 12.5 (7 patients). The acute case infers that brucellosis infection is very active and primarily the cause of the illness. One acute case terminated fatally. The man had an enlarged liver and a grave anæmia, which by the history antedated the acute superimposed Brucella infection. In this case dual pathological states were strongly suspected. Death came rapidly from increasing septicæmia and terminal peritonitis. Autopsy was not granted. One patient had recurrent mildly acute exacerbations at long intervals. The balance, 5 patients, recovered fully, clinically, but not entirely, serologically. It is of interest that of the 7 acute forms 3 had only general systemic febrile manifestations plus positive blood findings, and the remaining 4 had each as well, (1) a massive pelvic abscess due to a ruptured appendix subsequently removed; (2) an acute abdominal condition, possibly surgical, treated expectantly because of both fever and strong agglutination reaction with recovery; (3) physical and x-ray left pulmonary apical disease which cleared entirely after the initial febrile state subsided; (4) enlarged liver and anæmia complicating the fatal case. Obviously an epidemic was not experienced so far.

There is nothing characteristic about the acute form except in the entirely uncomplicated case, when it can be observed that the patient perspires intensely and claims he does not feel hot although he registers a high fever. Actually at the height of the disease the patients did not appear as sick as might be judged by the fever and progressive debility. By referring to Table I it will be seen that fever was listed in all cases; sweats, chills, loss of weight stand alike in frequency next; then came headaches, emesis and abdominal pain in the same number; one each of hæmoptysis, vertigo and joint pains occurred. Finally, the table indicates that independent complications do not preclude the presence of acute brucellosis, as 4 out of the 7, including one fatality, are in this category.

TABLE I.
SEVEN ACUTE FORMS

Case Number	*1*	*2*	*3*	*4*	*5*	*6*	*7*
Fever	x	x	x	x	x	x	x
Sweats	x	x		x	x	x	
Chills	x	x		x	x	x	
Loss of weight	x			x	x	x	x
Headache		x		x	x		x
Emesis			x	x			x
Abdominal pain			x	x	x		
Hæmoptysis							x
Vertigo						x	
Joint pains						x	
Complications			x	x	x		x
Positive serum	x	x	x	x	x	x	x
WBC+−N	−	N	+	−	+	−	+

Vertical = Signs in each case.
Horizontal = Frequency of stated signs.

Chronic cases.—Fifty cases were chronic. One might say that the surviving acute ones, except one recurring case, are probably now also chronic. If so, they are not in need of any special attention. In the chronic case, that is in the person who is not well, who has certain types of complaints and positive serological findings, there are three questions to answer. Is the infection the sole disability? Is it the cause of the pathological state which alone can

account for the complaints? Or is the infection an incidental complication?

A survey of the chronic cases suggests a possible grouping. This is useful, because there is no all-embracing symptom-complex that would justly describe individually chronic forms. There is however a good guide when 10 cases belong to a group who have clinically almost entirely subjective complaints of a neuropathic type, 15 are strictly undetermined general or constitutional debilities, 16 have other demonstrable diseases of which one type is especially prominent, but brucellosis is also known to be the cause of many such pathological changes, and, lastly, of 9 cases 6 are proved to be suffering from well-established fully accounted-for sickness, and 3 healthy persons in whom the finding of the infection is perhaps at best only a complicating incident. Table II further illustrates sub-groups of well

TABLE II.
FIFTY CHRONIC CASES

GROUP I=10		GROUP II=15	
Neuropathic		*Systemic debilities*	
Impotence	3	Loss of weight	9
Exhaustion	3	Indeterminate indispositions	6
Neuritis	2		
Cephalalgia	2		
Hypoglycæmic syncope	1		
GROUP III=16		**GROUP IV=9**	
Combined with		*Incidental to*	
Arthritis	8	Contact	3
Arteriovascular	2	Parkinsonism	2
Cholecystic and gastric	3	Syphilis	2
Phlebitis	2	Diabetes	1
Iritis	1	Tuberculosis	1

known clinical entities where brucellosis offers etiological possibilities. Of all the cases considered four hold especial interest. Two patients have Parkinsonism. One at 45 years of age has not the usual preceding history. The other is even more striking, as he conforms clinically with a diagnosis of chronic brucellosis, his serum reaction is positive, but the neurological aspects are definitely those of chronic encephalitis and Parkinsonism. Two are of immense importance medico-legally. The first of these a middle-aged woman was struck by a street car. She continued to complain of headaches, nervousness, pain in the right shoulder, arm, also in the right ankle and numbness on the top of her head over a greater area than the original scalp wound. It was elicited that she had "chills". She appeared myxœdematous; the basal metabolic rate was minus 26. The blood uric acid, ascertained in relation to an arthritic factor, was 6.4 mg. per 100 c.c. of blood. Her blood agglutination for *Br. abortus* at the time of examination for legal discovery was positive (1/100), and immediately prior to the date of the trial had increased in intensity and a positive result was obtained at an even greater dilution. All other findings were subjective and mainly matters of opinion. The defence plea sponsored the modern concept regarding brucellosis but failed to impress the court because medical evidence was of necessity in this disease more vague than factual. Out of this experience arise two things of note. First, the court insists on more unanimity of opinion and we require more knowledge of this disease; secondly, trauma can predispose to exacerbations. Another case here included is pending court procedure. Again, arguments on both sides will probably leave the court bewildered.

COURSE AND OBSERVATIONS

Concrete evidence is lacking as to how long infection might have existed in those cases classed as chronic. The history in one case of general debility dates back twelve years. There is no satisfying means by which to predict the continuation of an infection. This latter consideration depends largely on what standard is adopted for cure. The best guide so far is the patient.

A doubt is expressed as to whether the question under discussion really exists at all as a problem, either in diagnosis or as a morbidity factor. The challenge does not appear in writing though it is often boldly spoken. On close acquaintance with the subject, especially after practical experience, one is definitely impressed. Amongst other things it is curious that veterinarians as a group are more often involved than any other class. Then, in many instances we have been able to trace and identify later infection in a herd where the source of milk supply was strictly known.

The Health Department and Ministry of Public Health of Saskatchewan have been vitally interested and particularly cooperative. The disease is reportable in the Province. While this regulation may be good for cattle the justification is not complete as to its pur-

posefulness in the human, except as a means to trace the source of infection. It is not wise to tell a perfectly healthy person with a positive agglutination test that what he is living with on apparently harmonious terms is after all a disease. Every case must be judged by all individual details. So far, it is altogether likely that the healthy person with infection is a prospective candidate for breakdowns which will in many instances be called "flu", overwork, exhaustion, etc. Whatever the process may be, the chronically afflicted demonstrate that brucellosis is an undermining influence. The relatively high incidence of arthralgias cannot be conceived of as entirely accidental. Likewise in neurasthenia Brucellosis should be considered as another possible cause, but making it a case of *prima facie* evidence is a contradiction to proved traditions. When this infection is the basis of a neurasthenic complex revealing the true cause to the patient is a potent influence in re-adjustment; that improvement is not yet due to any other form of specific treatment at our command. It has the reverse effect if we stigmatize an innocent person erroneously.

TREATMENT

A review of all the tried means reported in the literature will not be attempted here. In our cases no original treatment was undertaken. Some generalities will be summed up briefly. In all acute stages feeding was plentiful and even forced; there were no restrictions because of fever. Antipyretics and antiseptics have no effect, but analgesics help to tide the crest. The routine was more along typhoid fever lines. A selected few of the chronic variety who had the convenience received vaccines. A remarkable instance occurred as the result of an order that miscarried. A graduated subcutaneous dose was given intravenously by mistake to one with painful arthritic joints who had a strong *Br. abortus* agglutination. The consequence was a rise to 105° F. temperature, with shock, and followed by very evident rapid improvement of her complaints where everything tried previously failed. The response of course is not unlike non-specific protein shock. In the case with iritis an extreme sensitivity existed, for the ordinary graduated subcutaneous doses produced reactions not anticipated. At the time such reactions were aggravating but eventually good resulted. Nothing spectacular or obviously creditable was observed in others receiving vaccine in whom a reaction was not produced. After the administration of vaccine blood titre tests were always increased, though there was no noticeable aggravation otherwise. This is to be borne in mind or false conclusions may be drawn from blood tests. For some reason, even in the case of the already infected individual, a change of milk supply was advised. This appears to be worth while. If correct, it is because either added numbers or a continuous supply of organisms severely taxes the immunizing capacity.

CONCLUSIONS

Brucellosis infection is prevalent. A correlation is necessary between laboratory reports of any test and the individual case. Positive blood reaction tests are common. The final diagnosis, irrespectively, rests on combined evidence with the clinical findings. At all times bacteriological evidence is significant of past disease, or future potentiality, and is not always the sole cause of present disorder. In the present, infection may be either acute or chronic. As an acute infection it is always the primary disabling factor. In 43 per cent of the acute cases only general systemic manifestations are found; in the others brucellosis should be thought of in combination with atypical or spurious phenomena, as, for example, in the case with overwhelming general debility and a massive pelvic abscess, which is not exactly a familiar combination. The chronic infections are by far the commonest and the diagnosis is even more elusive. Clinical groups are cited which seem to show a special predilection for occurrence of the infection. This knowledge serves as a clue to the more frequent discovery of the offending condition.

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INSTITUTIONAL CARE IN THE TREATMENT OF POLIOMYELITIS*

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IT is the intention in this paper to give an outline of the pre-surgical after-treatment of poliomyelitis as is carried out on the wards of the University of Alberta Hospital. These patients are admitted from the isolation hospital or as soon as they are released from quarantine in their homes. This form of treatment precedes by a long time any surgical intervention, and can be instituted at the earliest possible moment. In fact it should be begun as soon as the diagnosis is made. The idea of starting this treatment so early is entirely one of conservation of resources, and is designed to prevent further loss of power in the affected muscles by over-use and by allowing them to be stretched. When we have reached the convalescent stage we endeavour by physiotherapy to increase the strength in these muscles and to bring them back to as nearly normal as possible. The greater the amount of muscle salvage, the better the end-result, whether the patient has to undergo reconstructive surgery, or whether such surgical procedures are considered unnecessary owing to the degree of recovery.

For purposes of discussion and treatment the course of a case of infantile paralysis is divided into three stages. These are—the acute, the convalescent, and the chronic. The acute stage lasts from the onset until the disappearance of the tenderness, usually from four to six weeks, but it may be prolonged by improper treatment. From its duration, part of its course is intra-quarantine. When the tenderness has departed we enter the convalescent stage, which persists for two years. This somewhat arbitrary period is laid down because all cases improve spontaneously, to a greater or lesser extent, for a period of about two years. When that time has been reached we can expect no further advance, and then we enter the chronic, which persists throughout life. The treatment in the acute stage consists of rest, prevention of deformity, and the avoidance of meddlesome therapeutics. In the convalescent stage the mainstay of the treatment is muscle training, and surgery is only indicated to remove some deformity which prevents the carrying out of the physiotherapy. In the chronic stage the treatment is principally surgical, with a certain amount of post-operative muscle training to improve function.

As pointed out above, the treatment in the acute stage comes under three headings—rest, prevention of deformity, and the avoidance of meddlesome therapeutics. By rest we mean as close an approximation to the dictionary definition of the term as is possible. In children it is not sufficient to order the treatment, but we must employ some means of seeing that it is carried out. This is best done by the use of Bradford frames. In adults the Bradford frame is used in cases of wide-spread paralysis or when the back and abdominal muscles are involved. This apparatus allows all the nursing procedures to be carried out with the smallest expenditure of energy on the part of the patient. It adds greatly to the comfort of the adult to use a small pillow to support the lumbar lordosis. This is not necessary in children, as the lumbar curve does not appear until five years of age and these young patients are very adaptable to any form of apparatus. The knees should be supported by a pillow so that they are one or two degrees short of full extension. This relieves the joint from all strain and prevents the stiffness so commonly seen in joints which have undergone a period of fixation in a position of strain. Patients with involvement of the arms and pharyngeal muscles will, of course, have to be fed. While talking of rest, I would like to condemn a common practice. This is the continued endeavour to elicit the reflexes whenever the patient is visited and also the repeated testing of the muscles by holding up the part and letting it drop. Once the diagnosis has been made, the rest treatment should be enforced and these examinations reduced to the necessary minimum.

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If deformities are allowed to occur they must be removed before active treatment can be undertaken. This takes time, and in many cases the patient has to be subjected to some form of operative surgery. Deformities weaken muscles by stretching them and not allowing them to contract. Flexion deformities are the forms commonly seen in the paralyzed patient and leave him in a very helpless condition. With flexed hips, flexed knees, the patient is only good for sitting or lying, but is no good for walking. It should be our aim throughout treatment to prevent any of these flexion deformities from occurring and to keep the patient in the same position that he is when standing. The easiest method of maintaining this position is by keeping him in the horizontal position with a minimum of sitting up.

Apparatus for the extremities consists of light splints or plaster casts. Our experience has been that there is less wear and tear on the hospital bed linen by using the bivalved plasters than with the metal splints. Drop-foot appliances should come up well past the end of the toes in order to prevent toe-drop, which is quite disabling. Drop-wrist splints should not hold the fingers in a position of strain in hyper-extension, so commonly seen, but should allow a certain amount of flexion to occur, which is much more comfortable. No very neat or effective apparatus has been designed to hold the thumb in opposition in the case of paralysis of the opponens pollicis. Paralysis of this muscle gives a flat hand, which results in a fairly large disability and is very difficult to relieve. Abduction splints for paralysis of the deltoid keep the arm at right angles to the body. The older models kept the arm in the coronal plane but the newer ones have the arm carried forwar 20 or 30 degrees, which is much more comfortable. Involved abdominal muscles are protected by the use of a many-tailed binder. This makes a well fitting surgical belt extending from the symphysis to the umbilicus, and is prevented from slipping up by using the lower strips of the binder on each side as perineal straps.

Avoidance of meddlesome therapeutics is a nice-sounding term, but its carrying out is sometimes difficult. By carrying out rest treatment we appear to the parents and friends to be doing nothing, and to their manner of thinking the treatment of the patient demands action and lots of it. In the early stages of this disease rest is the treatment *par excellence*. The serum treatment, to be of any use, must be given early and long before the case would come to our wards. The tenderness is best treated by hot packs. Morphia may be necessary at times, but absolute rest on a Bradford frame, coupled with hot packs, is usually sufficient. The paralyzed muscles must be kept warm, as we find there is a disturbance in circulation.

When the tenderness is gone, we enter the convalescent stage, and absolute rest is replaced by an increasing degree of active treatment. The first thing to be done is a muscle test, and every muscle or group of muscles has its power tested and charted. No instruments of precision are used, but the test is a manual one carried out by the physiotherapy staff. The muscles are rated as normal, good, fair, poor, or trace. The last is charted when the muscle belly is felt to quiver but there is no action in the part. A poor muscle is one that can move the part but cannot lift it against the action of gravity. A fair one can move the part against gravity but against no added resistance. A muscle is rated as good when it can move the part and against considerable added resistance. A normal muscle is one which is considered to have the right amount of power or is equal to the strength of the unaffected fellow of the opposite side. When the muscle test is completed we have a picture of the case on paper and are ready to begin our active treatment. Muscle tests are taken every three months and the progress noted. It is felt that this is frequent enough, as sometimes the changes are so slight that a more frequent examination would fail to disclose them.

The idea of this treatment is to increase the power in the affected muscles, to bring poor ones up to fair and on to normal, if possible. Between these treatments, the paralyzed muscles are of course replaced in their protective apparatus to prevent stretching. In some cases after the commencement of this active treatment, we get a return of the pain and tenderness in the muscles, and of course then the patient must return to the regimen of absolute rest for a while. This occurred quite frequently among our 1935 cases—the tenderness reappeared on the institution of physiotherapy.

Physiotherapy may be discussed under four headings—heat, massage, electricity, and muscle training or muscle re-education. All muscles work better when they are warm. The baseball managers know this, for before a change of pitchers during a ball game there is always a period of warming up for the incoming performer. Massage increases the circulation of the blood through the paralyzed muscle and thereby keeps up its nutrition. Before massaging the muscles are warmed up by dry heat, applied through the medium of the electric light heat can. Electricity is used by a number of schools to build up the muscles. The great disadvantage is that if any of our patients of tender years receive a shock they lose confidence in the treatment, and if treatment is to be successful we must have the confidence of the patient. We feel, too, that it is much harder to gauge the onset of fatigue by this method than by muscle training; therefore, we do not use electricity. By far the greatest progress is made by what is known as muscle training or muscle re-education. An athlete entering a competition trains for the event by increasing the power of his muscles, so that in the case of a foot race he is enabled to run faster than before training. The same idea is applied to these affected muscles. By having the patient practise contracting the weakened muscle we hope to increase the power in that muscle. But at no time must we allow fatigue to show up. If this is not carefully watched for we may change a partial paralysis into a complete one. If the fourth contraction of a muscle is not so good as the third, then fatigue is present and we must stop. The extremity is then put back in its protecting splint and rested until the following day, when a new attempt will be made. This is repeated day after day until the muscle that appeared to be gone shows a slight return of power. This is carefully nursed along, in the hope that we will get the muscle to be strong enough to be of some use.

From this description it will be seen that this work must be carried out by highly skilled and well trained workers. They must have a good working knowledge of anatomy, so that they exercise the correct muscle and allow no trick movements to be done. When these are not noticed and allowed to occur we are then training the wrong muscle. As the progress in many cases is very slow these members of the staff must be endowed with infinite patience and tact. Sometimes during the muscle training the progress of these patients seems to come to a standstill. When this happens it has been our practice to give them a change of scenery, and send them home for a month or two and they return with renewed interest in their treatment.

No mention of physiotherapy is complete in these times without saying something about the underwater or pool treatment. These pools are found in most institutions and vary from the tiled swimming pool to the sheet metal tank resembling the old fashioned bath tub, which accommodates one patient at a time. The patients are put in the tank and carry out their muscle training in the water. By this method they gain the extra support of the water in moving the part. Fatigue does not appear so readily by this means, and consequently we can carry out more muscle training. One point in particular should be noted with this form of treatment and that is the wonderful effect it has on the patients' morale. It peps them up and they seem to have a new lease on life and take a greater interest in their treatment. It must be remembered that muscle training is not very effective in a stale patient.

As the muscles increase in power under treatment the patient is allowed more freedom, and may be allowed out of his apparatus an increasing amount of time each day. In allowing patients to sit up the abdominal muscles and back muscles must be carefully watched and supported by a binder, or be well pillowed up. Any increase of activity on the part of these patients must be started with very small amounts and gradually increased. Probably the best method of measuring the amount of activity allowed is timing by the clock. The next question that comes up, of course, is when are we to allow these people to get up and about? Lovett laid down some years ago that walking should be prohibited for a year, and patients were allowed up before this only if they had made a complete recovery or if it was thought that no further progress would be made. We use the above rule as a guide only, and usually are able to carry it out fairly well in the case of children, but with adults there are so many factors to be considered that each case must be worked out on its own merits.

Whatever rule we adopt, when we first get these patients up after a prolonged rest we must protect the weakened muscles by some form of apparatus. Any such apparatus must be comfortable, light and effective.

When the termination of the convalescent stage is reached, the chronic stage begins. During this period we do not expect to gain any further power in the affected muscles. Any further progress is made by reconstructive surgery. Before embarking on operative surgery we must be sure that muscle progress is at a standstill. For example, a valgus foot, due to the paralysis of the tibialis anticus, can be improved by transplanting the tendon of an active peroneus longus. If this is done too early and the tibialis anticus regains its power, then we have two strong muscles pulling the foot into varus, which is just as bad a deformity as the original valgus. Following these operations there is usually a short course of physiotherapy to restore function. Reconstructive surgery at the present time is more extensively carried out than in the past, to relieve these patients from carrying around the various pieces of apparatus which used to be so fashionable.

The foregoing is an outline of the early after-treatment of infantile paralysis. It may be called the non-surgical treatment, and a great deal may be accomplished by it. This is not decrying the surgical treatment. That has a very important place in the after-treatment, but like many other things there is a proper time for it.

OUR EXPERIENCE WITH THE HORMONE TREATMENT OF THE ADENOMATOUS PROSTATE*

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THE following report is based on the results obtained in the Urological Department of the Toronto General Hospital, and it has been carried out in collaboration with Drs. Lower and McCullagh of the Cleveland Clinic. Twenty-one cases are reported, although several others were observed. The treatment is based generally on the work of various investigators who have proved the existence of and synthetically prepared at least one particular hormone (Testosterone) which has an effect on the secondary sex glands. More particularly however, this study has to do with the theory of McCullagh, of the Cleveland Clinic, based on experimental studies, who believes there are two testicular hormones—one formed by the tubular or germinal cells which he calls "inhibin", the function of which is to act as a control to the pituitary gland; the other, "androtin", produced by the interstitial cells and acting as a sex stimulant, which activates and causes the prostate to hypertrophy. The production of androtin is in turn regulated by the anterior pituitary. Inhibin has not as yet been isolated or assayed, but as the result of experiments it is concluded that it is water-soluble and absorbed by the stomach, and can therefore be successfully administered by the mouth. Androtin has been found in the blood, spinal fluid and urine. It has been isolated, assayed, prepared synthetically, sold commercially, and is administered hypodermically.

A short cut may here be taken by quoting a few pertinent experiments on which inhibin treatment is based.

1. The castration of male rats results in changes which can be placed in two distinct groups, namely, (*a*) those in which secondary sex characteristics are lost and the secondary sex glands (prostate and seminal vesicles) atrophy, and (*b*) those in which the pituitary gland undergoes hypertrophy and becomes hyperfunctional; the adrenals also hypertrophy.

2. Testicular extracts prepared with fat solvents will prevent the atrophy of the secondary sex glands if administered immediately after castration. If the glands are permitted to atrophy the testicular extracts will cause regeneration.

3. The comb-growth-promoting substance from male urine has the same physiological properties as the hormone extracted from testes. The known chemical and physical characteristics of the hormone from testes and that from urine would indicate that they are identical.

4. The hormone from urine or blood is derived from the testes, since it is not found in the body fluids of castrated men, although it is readily demonstrable in normal men.

5. Such a hormonal preparation from urine, when given in doses sufficiently large to cause regeneration of the atrophic secondary sex glands of castrated rats will

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neither prevent nor correct the hypertrophy of the pituitary gland and adrenals after castration.

6. Aqueous testicular extracts, which could have contained no more than an insignificant amount of the prostate-regenerating hormone, prevent the cellular changes from appearing in the pituitary gland after castration of rats, and also completely inhibit the hyperfunction of the pituitary gland.

7. Destruction of the germinal epithelium of the testes will cause pituitary hyperfunction without causing atrophy of the secondary sex glands.

The apparent and only obvious conclusion is that the testicle secretes a hitherto unrecognized water-soluble hormone, one function of which is control of the pituitary gland.

To facilitate discussion it is essential that the two testicular hormones be differentiated. Using the root of the Greek word ἄνδρος the name "androtin" has been suggested for the benezene-soluble substance which is responsible for the development and maintenance of the secondary sex glands and other secondary sex characteristics. This name corresponds to "theelin" suggested by Doisy for the ovarian hormone. The water-soluble testicular factor, which is characterized by its action on the pituitary gland has been called "inhibin", from the Latin word *inhibere*.

The arguments in favour of the presence of two testicular hormones are almost, but not absolutely, conclusive. It has been demonstrated that "inhibin" in doses which do not influence the secondary sex glands will prevent the hyperfunction of the pituitary gland of castrated animals. Also, as mentioned above, "androtin" in doses sufficient to cause regeneration of the atrophic prostate of castrated

Table I.

Inhibin Only

Name	*Symptoms*	*Treatment*	*Remarks*
Cavanaugh Age 60 Adm. Apr. 8/35 Dis. June 8/35	Difficulty and slow stream 1½ years. Retention 1½ years ago (occas.). N.F.—2-4 / D.F. q.½.h. for 6 mos. Bladder to umbilicus. Left infected hydronephrosis.	Apr. 8—Decompression. Apr. 8-15—Retention catheter. Apr. 15—Catheter removed R.U. ozs. 1½ to ozs. 8 for 5 days without Inhibin, daily tests. Apr. 16—Reinserted. *Inhibin started.* May 5—Epididymitis — catheter removed. R.U. oz. ½—free, good stream. June 8—R.U. none. Home.	Urethrogram No. 42950. Biopsy — Chr. Prostatis. Cystos.—Bi-lobe. NPN on admission 48; discharge 41. Number days treatment before able to void satisfactorily 19. On discharge R.U. none—stream free. June 2 — O.P.D. N.F.—0 / D.F.—5 R.U. ozs. 3, feels fine. *Impression:* Much improved.
Plested Age 65 Adm. Oct. 30/34 Dis. Apr. 18/35	Increased D. & N.F. for 1½ years. Retention and catheter drainage for 5 weeks in another hospital. Has goitre.	Oct. 30/34—Catheter drainage. Dec. 7—Thyroidectomy—rocky time—still on catheter drainage. Jan. 26/35. Catheter removed —could not void. Jan. 26/35—*Started Inhibin* 3 p.c. Feb. 28—R.U. ozs. 8 (one month). Mar. 15—R.U. ozs. 8. Apr. 14—R.U. none. N.F.—9 / D.F.—3	Catheter drainage alone 17 weeks. Treated with Inhibin 73 days. Saw him 2 months later at home. R.U. none—normal function. Cystos.—Bi-lobe. Biopsy—Chr. Prostatis. Recent letter states he is well. *Impression:* Much improved.
Benson Age 68 Adm. May 8/35 Dis. June 30/35	Dribbling for years. Diff. 1 year. Small stream 3 months. Retention 6 days.	May 8-30—Catheter drainage. May 30—Catheter removed R.U. ozs. 2 without Inhibin. May 30—*Started Inhibin* 3 p.c. and h.s. June 10—Catheter removed R.U. oz. 1. Interval catheterization R.U. oz. 1. June 30—Discharged R.U. oz. 1.	Bi-lobe. Very large. Twenty-two days drainage reduced R.U. to oz. 2. June 30—R.U. oz. 1 free. June 2/36—O.P.D. N.F.—0 / D.F.—6 —clear, free. R.U. oz. 1½ Prostate quite small. *Impression:* Much improved—wants work.
Carr Age 55 Adm. Apr. 12/36	Frequency — over distension. Infected bilateral hydronephrosis. NPN 135—Other tests poor.	Decompression drainage. Apr. 28—*Inhibin started.* May 20—Nauseated — discontinued no effect. NPN 135. Two c.c. Cortin B.D. NPN reduced to 50.	Bi-lobe. Inhibin—no effect. Cortin reduced NPN but did not improve kidney function. *Inhibin failure.*

rats will not prevent the hypertrophy and hyperplasia of the pituitary gland.

The hypothesis of the duality of testicular endocrine function has been developed to explain well-controlled animal experiments. In addition to this experimental evidence there are certain general considerations which lead to the belief that there might be two hormones secreted by the male gonads. The testicle is the male analogue of the ovary; androtin is comparable physiologically and chemically to theelin. Until now there has been no suggestion of a testicular hormone comparable to ovarian "progestin". The idea that the testicle produces two hormones is compatible with the histology of the gland.

Prostatic hypertrophy, as observed clinically in many adults beyond middle age, has had no satisfactory explanation. It is now a well-recognized fact that the pituitary gland can stimulate the testes to the production of sufficient androtin to cause prostatic hypertrophy in rats. If the testicular cells producing inhibin were to fail, previous to the failure of those structures which produce androtin, the hypertrophic phenomenon could easily be explained. The absence of inhibin would result in hyperfunction of the pituitary gland, which, as pointed out above, is a known cause of prostatic hypertrophy in rats. The brilliant researches of Martins and Rocha indicate that there is every

TABLE II.

INHIBIN ONLY

Name	*Symptoms*	*Treatment*	*Remarks*
Lennon Age 86 Adm. Oct. 12/35 Dis. Nov. 14/35	Catheterized 4 times last 2 years for retention. N.F.—3 Slow stream D.F.—4 and difficulty Infected hydronephrosis.	Oct. 12-29—Catheter drainage. Oct. 29—Could not void. *Started Inhibin* 3 p.c. Nov. 8—Catheter removed R.U. oz. 1. Nov. 12—Voiding frequently but freely. R.U. oz. 1. N.F.—5-6 D.F. q.1—2 hours Nov. 14—Discharged R.U. oz. 1. May 8/36—O.P.D. R.U. 0 N.F.—3 D.F. q.4.h.	NPN on admission 36; discharge 36. Cystos.—Bi-lobe. Number days treated before able to void, 11. June 2/36—O.P.D. N.F.—3 D.F. q.4.h. R.U. oz. 1. *Impression:* Much improved
Millard Age 83 Adm. Sept. 6/35 Dis. Oct. 17/35	Difficulty 2 years. Dribbling N.F.—4-5 D.F. q.1.h. Sudden retention. Temperature 102°. Pyelonephritis.	Catheter drainage. Catheter removed Sept. 20—retention. Sept. 20—*Inhibin started* 3 p.c. Oct. 4—Catheter removed. R.U. ozs. 2. Oct. 17—Discharged. R.U. oz. 1.	Biopsy none. Cystos. Bi-lobe. Catheter drainage 2 weeks did not improve him. Took Inhibin at home till Nov. 30th. Jan. 3/36—O.P.D. R.U. oz. 1. N.F.—2-3 D.F. q.4.h. voids freely. June 2—O.P.D. N.F.—3 D.F.—5 R.U. oz. ½ cloudy. Rect. Prostate not much enlarged if any. *Impression:* Much improved.
Wilson Age 46 Adm. Apr. 5/36 Dis. Apr. 6/36	Frequency only. N.F.—3-4 D.F. q.2.h. urine cloudy. R.U. ozs. 14. Cystos. negative for obstruction. Wassermann, negative. Neurological signs negative.	No drainage. Daily catheterization by own doctor for 6 weeks. June 25/35—R.U. ozs. 13 and *started on Inhibin* 3 p.c. till Aug. 26/35. R.U. *none* and urine clear. Frequency disappeared. Mar. 31/36—R.U. ozs. 4, cloudy. Inhibin started again.	Given to determine effect on atonic bladder. June 2—Has had Inhibin for last 2 months. June 2—R.U. oz. 1. N.F.—0 D.F.—4 —no Inhibin for 2 weeks. *Impression:* Much improved.
Church Age 69 Adm. Apr. 27/36 Dis. May 30/36	Difficulty—slow stream. N.F.—2-3 D.F. q.2.h. Retention on admission. Large prostate per rectum.	Catheter drainage. Apr. 29—*Inhibin started.* May 20—Biopsy. May 21—Bladder spasms without cause. May 23—Catheter removed. Pt. voided—no residual. Spasms gradually subsided also frequency. Progressively better till discharge.	Cystos. — Bi-lobe — large. Biopsy—Adenoma 2 pieces. On discharge N.F.—1 D.F.—6 free stream. Very happy. *Impression:* Much improved. Do not know how much good Inhibin did.

possibility that inhibin is not produced when the germinal epithelium is destroyed. The destruction of the germinal epithelium does not change the secondary sex characteristics which are maintained by androtin. It will thus been seen that it is reasonable to suppose that prostatic hypertrophy is caused by a deficiency of one hormone, inhibin, and a consequent increase in another, androtin. The results herewith reported are those which we obtained from the administration of desiccated testes, given with the idea that we were at least helping to supply the deficiency.

Concurrently with this work we tried a few experiments in ligating the vasa efferentia. These were done last winter with varying results. The theory here is that testicular hormone is retained under the tunica albuginea and is re-absorbed by blood vessels. Another theory is that the ligation stimulates a number of nerve ganglia surrounding these tubules. We incline to the former view because in one of our successful cases the tubules were accidentally divided. In any event, although our cases were very few, our results did not seem to correspond with those of Niehans, published in *The Lancet,* February 8, 1936. However, there were certain observations which led us to believe that some change takes place. For instance, in our successful cases which were assisted by inhibin there was marked frequency and bladder spasm. The Tables represent the hospital histories of 18 cases which were started in the fall of 1934. Three others are also appended, including 2 of Dr. Fould's at St. Michael's, (one of which was an inhibin failure, and the other a successful ligation); the third was one of our own ligation cases, classed as an improvement because he voided freely in three days with 2 oz. of residual urine. In ten days the residual urine had diminished to one-half an ounce but the frequency and bladder spasms were so great that we were forced to drain the bladder and remove the prostate a month after the ligation. Several other cases were unsuccessfully treated with inhibin at

TABLE III.

INHIBIN ONLY

Name	*Symptoms*	*Treatment*	*Remarks*
Irvin Age 78 Adm. Nov./35 Dis. Feb./36	Retention. Bronchial asthma. Heart bloc. Diabetic and uræmic. Coma NPN 138. Blood sugar 0.44 plus.	Catheter drainage. Intravenous and Insulin. Dec. 10—*Started Inhibin* 3 p.c. Jan. 17/36—No effect. Suprapubic prostatectomy.	Mostly median lobe hypertrophy. Died 4 months after leaving hospital. *Impression:* Failure.
Thompson Age 82 Adm. Jan. 28/36 Dis. Feb. 18/36	Had been admitted on Dec. 17/35 with chr. retention for 6 months.	Dec. 25/35—*Started Inhibin.* Jan. 25/36—No result. Jan. 28—Enucleation.	Tri-lobe. *Impression:* Failure.
Strangway Age 72	Chr. retention—R.U. ozs. 30 with median bar.	Decompression and drainage. Transurethral resection followed by R.U. ozs. 15. This continued with irrigations for 9 months. R.U. ozs. 15. Inhibin 3 p.c. for 3 months—no effect.	Very atonic bladder. *Impression:* Failure.
Jones Age 68 Adm. Apr. 18/36 Still in hospital	Difficulty and frequency for 2 years. Infection—pyelonephritis—diabetes.	Decompression — drainage, Med. ward for diabetes. Apr. 19 to May 18—on *Inhibin.* No effect. Perineal prostatectomy.	Tri-lobe. *Impression:* Failure.
Beatty Age 68	Chr. retention—ozs. 80. No enlargement of prostate. Wassermann, negative. Neurological symptoms negative. Suspect—spinal sclerosis.	Decompression and drainage. Drainage only for 1 month and R.U. ozs. 80 still. Dec. 18/35—*Placed on Inhibin.* Jan. 17/36—R.U. ozs. 34. Jan. 20/36—Suprapubic.	Inhibin given here to see if it would have any effect on bladder tone. There appeared to be some effect but patient was discouraged and wanted to get home. *Impression:* Showed signs of improvement. Is taking it again recently.

home without drainage by all the members of our Staff, but no accurate records were recorded.

In the 21 cases reported the average age was 70.8 years. The youngest patient was 46 and the oldest was 86. Seventeen patients were treated with inhibin, with 10 failures and 7 improvements. At least two of these (Beatty and Strangway) should not be included, as both had absolute loss of bladder tone and were only given inhibin out of curiosity. Beatty had spinal sclerosis and no obstruction whatever. That really leaves 46.6 per cent improvements and 53.3 per cent failures. In the improved cases the average number of days on inhibin, before we were able to permanently remove the catheter, was 31, the longest period being 78 days and the shortest 9 days. As to

TABLE IV.

INHIBIN AND LIGATION

Name	*Symptoms*	*Treatment*	*Remarks*
Tucker Age 79 Adm. Jan. 19/36 Dis. Mar. 27/36	Very rocky old man. N.F.—3-4 D.F.—5-6 years. Daily catheter by own doctor for 1 week. Bi-lobe.	Catheter drainage, 17 days. Feb. 5/36—*Inhibin.* Feb. 17—Catheter out; voids easily but frequently q.1.h. R.U. oz. 1. N.F.—4 D.F. q.1.h. Feb. 25—Ligation. Inhibin discontinued—voiding freely. Mar. 27—On discharge R.U. oz. ½ N.F.—2-3 D.F. q.2.h. Feels fine—elated with result.	June 2—O.P.D. Wonderful change for better in appearance and actions. Voids freely N.F.—3 D.F.—5-6 R.U. ozs. 2 cloudy—++. Rectal—Moderate enlargement. *Impression:* Much improved.
Baillie Age 67 Adm. Dec. 31/35 Dis. Feb. 11/36	Difficulty 2 years with multiple retentions. N.F.—1-2 D.F.—6-7 retention huge prostate. Bi-lobe.	Jan. 6/36—Inhibin. Jan. 12—Could not void. Jan. 13—Ligation. Jan. 18—voids with ease q.2.h. R.U. ozs. 8. Jan. 21-30—Catheter re-inserted. Feb. 1—Voiding—feels fine—R.U. ozs. 2. Feb. 3—R.U. ozs. 3 clear. Feb. 11—Home. Mar. 3—O.P.D. R.U. oz. 1. N.F. occ. 1 D.F.—4-5 free June 2—O.P.D. N.F.—1 D.F. q.2.h. R.U. ozs. 2 cloudy. Rectal—Prostrate quite large still.	Very large prostate. Much improved but feel he should have more Inhibin. Effect of ligation is not diminishing size of gland very much.
Hull Age 71 Adm. Feb. 7/36 Dis. Apr. 11/36	Retention 2 weeks—previous difficulty. Infected.	Decompression. Feb. 7-24—Inhibin. Failure. Feb. 24—Ligation—failure. Mar. 16—Prostatectomy (per.)	Tri-lobe—very large. Failure.

TABLE V.

LIGATION ONLY

Name	*Symptoms*	*Treatment*	*Remarks*
Carroll Age 88 Adm. Feb. 29/36 Dis. Mar. 21/36	Chr. retention. Hospital 3 times last year. Retention catheter all this time. Enters for ligation.	Mar. 2—Ligation.	Extremely debilated. Absolute failure.
Garbutt Age 71 Adm. Feb. 26/36 Dis. Apr. 14/36	Increasing frequency for 2 years. N.F.—5-6 D.F. q.1.h. Retention to umbilicus. Poor functions.	Mar. 10/36—Ligation. Mar. 11—Voided with comfort but R.U. ozs. 30. Mar. 31—Transurethral with usual course. Apr. 14—Discharge R.U. ozs. 2.	Failure.

the successful cases, on account of our limited supply, we were unable to administer inhibin long enough to reduce the size of the gland. Clinically, there was no change in the size, either per rectum or cystoscopically, but the patients emptied their bladders and did so easily.

Four cases were treated by ligation only, with two failures and two improvements, or 50 per cent for each, the improvements being two of the three appended.

Actually, four cases were treated with inhibin and followed by ligation. The fourth case is that of Carr, who was ligated after these tables were made. At no time since his admission could we leave him without a catheter. Within twelve hours of his operation, and continuing to the present, he has voided freely with a good stream. Although the first residual urine check, two days after, showed 15 oz., the amount has diminished in three days to 6 oz. If this case is included as an improvement, and we think it should be, it makes 75 per cent of improvements and 25 per cent failures. Three cases at the present are being treated with androtin (Ciba), but so far without results.

Conclusions

1. We believe in the theory of the dual hormone control of the prostate and secondary sex glands.

2. So far as "inhibin" is concerned our results compare fairly well with those of Lower and McCullagh, of the Cleveland Clinic.

3. Clinically, there has been to date no diminution in the size of the gland, either per rectum or cystoscopically, but in spite of this the function in some cases markedly improves. Neither from theory nor experiments would one expect a reduction in size in such a short time.

4. With the inhibin treatment alone, in-patients were treated by continuous drainage or interval catheterization; out-patients usually had neither. The difference in the results in favour of the former were so great that we were forced to believe that preliminary drainage reduced the prostatic congestion and at the same time improved the bladder tone, thereby paving the way, so to speak, for the hormone action. The results of some cases, on the other hand, proved that it was not altogether the drainage.

5. Inhibin is not indicated in cancer, median bars, large median lobes or tri-lobes.

6. In many cases, particularly with the ligations, there were marked frequency and bladder spasms in spite of an empty bladder. An increased bladder tone would thus be suspected. This action may be analogous to the uterine contractions of parturition and abortion, which are supposedly induced by some ovarian hormone.

7. Until the second hormone (inhibin) is isolated and assayed we have no conception of the amount of dosage given or required.

8. In the case of Carr the result was so astonishing that we are now ready to believe this minor operation will have a very definite place, and particularly so in those cases which are beyond the hope of major surgery.

9. *Re* the administration of "cortin", prepared by Dr. McHenry in the Connaught Laboratory and given to Carr, it certainly reduced the non-protein nitrogen and made a different man of this patient, but reduction of the non-protein nitrogen was not accompanied by increased kidney function.

10. From the results which we have seen, it would appear that bladder muscle tone has as much (or more) to do with residual urine as the varying degrees of prostatic hypertrophy.

Prontosil Treatment of Erysipelas.—Minkenhof reviews the literature and records his observations on 35 cases of erysipelas at the Wilhelmina Hospital, Amsterdam, treated by prontosil. The drug was administered exclusively in the form of tablets, the dosage being as follows: first three days 0.3 gram seven times daily, second three days 0.3 gram five times daily, third three days 0.3 gram thrice daily, and last three days 0.15 gram thrice daily. Children in the first year of life were given 0.15 gram twice daily for the second three days, and so on. Children from 1 to 4 years usually had 0.9 gram daily for the first three days. The urine always assumed an orange-red colour for one to two hours after administration of the first doses, and on the following days a darker red, while several days elapsed after prontosil had been stopped before the urine assumed its normal colour. No really bad effects were observed. Comparison with 35 controls not treated by prontosil showed that the drug considerably shortened the duration of the disease, though it did not prevent the occurrence of complications. — *Nederl. Tijdschr. Geneesk.*, Nov. 21, 1936, p. 5197. Abs. in *Brit. M. J.*

SOME OBSERVATIONS ON GLYCINE METABOLISM IN PATIENTS SUFFERING FROM MYASTHENIA GRAVIS*

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THE rationale of the treatment of patients suffering from myasthenia gravis has its basis in biochemical studies of protein metabolism. Rosenthal,[1] in 1870, pointed out that there were disturbances in the excretion of creatinine. Using cases suffering from myopathic disease, Levene and Kristeller,[2] in 1909, showed that these patients had a low creatinine and high creatine excretion. In 1929, Brand, Harris, Sandberg and Ringer,[3] while studying the metabolism of various nitrogen-containing substances in patients with progressive muscular dystrophy, found that there was a definite increase in the creatine excretion when they were fed with glycine. In 1932, Thomas, Milhorat and Techner[4] repeated this work, but extended the experiments over a much longer period. They noticed that the glycine had a definite beneficial effect therapeutically in cases of muscular dystrophy. It is generally conceded now that cases of myasthenia gravis are helped the most (Boothby[5]).

When the use of glycine for cases of myasthenia gravis was suggested it was thought that gelatine, which contains 25 per cent glycine, should be as efficacious and much cheaper. Brand[6] and his co-workers have shown that gelatine increases the output of creatine when fed to a patient suffering from muscular dystrophy. Accordingly, it was decided to investigate this aspect of the problem. Forty-five grams of gelatine were administered to Case 1† for thirteen days in substitution for glycine. This had to be discontinued because the myasthenic manifestations increased. The same procedure was also tried in Case 3 for twenty-three days without clinical improvement. Both these patients had responded well to glycine. The failure of gelatine to relieve symptoms as effectively as glycine suggested the possibility that there was a defective breakdown of the compounds containing glycine in the gastro-intestinal tract.

Gastric juice was tested for its ability to digest gelatine, but it was found that both normal persons and patients with myasthenia had insufficient digestion of gelatine *in vitro* to be demonstrable by precipitation with sodium sulphate. We then tried duodenal juice and were able to demonstrate considerably more digestion in normal persons than occurred in cases of myasthenia gravis. The samples were collected after the patient fasted over night. In some of the cases a long time was required before the duodenal contents could be obtained. Evidence was accepted that duodenal contents had been obtained when the juice was markedly stained with bile.

The details of the technique are as follows. Into an Erlenmeyer flask were put 6 c.c. of duodenal juice, 10 c.c. of ammonia buffer pH 8.9, 20 c.c. of 10 per cent warm gelatine, and 3 drops of chloroform. A control flask contained 6 c.c. of the boiled duodenal juice and the other constituents mentioned above. Both flasks were placed in a water bath at 37.5° C. and incubated for twenty-four hours. The percentage total nitrogen was estimated by duplicate Kjeldahl determinations on the contents of the experimental flask. The amount of subproteose nitrogen was found by precipitation of the proteose and protein by a saturated solution of sodium sulphate (two-thirds of a gram of anhydrous sodium sulphate for each c.c. of fluid). This was allowed to stand for one-half hour in a bath at 37.5° C. and filtered in the bath.* Duplicate nitrogen estimations were made on the filtrates, control and experimental. The amount of digestion of the gela-

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The cases studied are those reported in Dr. Hyland's paper (Myasthenia gravis: results of treatment of six cases, *Canad. M. Ass. J.*, 1936, **35**: 372). The same case numbers are used, and for detailed accounts of the history reference may be made to that paper.

† See Hyland, H. H.: Myasthenia gravis: results of treatment in six cases, Case 1, *Canad. M. Ass. J., loc. cit.*

* It was felt that it would be unnecessary to filter this material at 32.75° C., at which point sodium sulphate has no water of crystallization, under the conditions of this experiment.

tine was taken to be the difference in the nitrogen estimations of the filtrates between the experimental and the control flasks.

The duodenal contents of a number of persons were tested for ability to digest gelatine. The results are indicated in Table I.

TABLE I.

THE PERCENTAGE OF DIGESTION OF GELATINE BY DUODENAL JUICE

1. IN NORMAL PERSONS

Date	*Sex*	*Total % N. of digest*	*Filtrate of digest N. %*	*Filtrate of control N. %*	*Digestion %*
16/8/33	M	1.22	0.80	0.46	28.0
16/8/33	F	1.13	0.83	0.44	34.5
19/9/33	M	1.27	0.66	0.50	12.6
19/9/33		1.84	1.10	0.50	24.5
14/9/33	F	1.23	0.84	0.49	28.4
15/10/33	M	1.28	0.79	0.51	21.9
15/1/35	M	1.22	0.80	0.45	28.6
16/1/35	M	1.29	0.92	0.47	34.9
21/1/35	M	1.20	0.70	0.45	20.8
23/1/35	M	1.27	1.00	0.57	33.8
				Average....	26.8

2. IN MYASTHENIC PATIENTS

	Case					
18/9/33	1	F	0.98	0.62	0.52	10.2
13/3/34			1.23	0.57	0.42	12.2
26/11/33	2	M	1.23	0.50	0.49	0.9
19/1/34			1.22	0.54	0.47	5.7
15/4/34			1.25	0.72	0.71	2.4
7/6/34			0.95	0.58	0.58	0.0
16/11/34			1.20	0.73	0.46	22.5
5/10/33	3	F	1.27	0.77	0.49	22.0
*9/3/34			1.17	0.68	0.44	20.5
8/9/35			1.19	0.96	0.79	14.2
	4†					
20/11/34	5		1.23	0.45	0.44	0.8
25/1/35	6		1.22	1.01	0.63	31.1
					Average....	11.9

*Seven months pregnant.

†Repeatedly attempted, but unable to get tube into duodenum.

The average digestion of the normal group is 26.8 per cent, and estimation in the cases with myasthenia gravis is 11.6 per cent. It is of interest that these cases should show a definite decrease in the digestion of glycine. Unfortunately, the cases of myasthenia gravis available were few, so that averaging these values is not a very justifiable procedure. Noteworthy too is the apparently normal digestion in one case of myasthenia gravis, namely, No. 6. This patient received no benefit from the use of glycine. One would like to know if the creatine and creatinine output in such individuals differs from those showing poor digestion; unfortunately, we did not estimate the creatine output in our cases.

Benzoic acid is a constituent of a number of foods, being especially high in some fruits. This substance when free in the body is definitely harmful, and so the organism neutralizes it by combining it with glycine to form hippuric acid. The source of glycine is thought to be of exogenous origin, namely, food like gelatine, etc., and also endogenous when the exogenous supply is reduced by synthesis of glycine from other amino acids. All investigators do not agree with the theory that the body can synthesize glycine. Some feel that there are stores in the body for glycine, such as cartilage, fibrous and elastic tissue, which can be called upon during need. The question of the glycine reserves in a case of myasthenia naturally presented itself.

We thought it advisable to study the output of hippuric acid in a case of myasthenia gravis. No. 1 was given a high carbohydrate-low protein diet, and the hippuric acid output in the urine was estimated for some time before giving benzoic acid, during the administration of 3.3 grams of benzoic acid (Merck), and after. A control case was also studied in a similar manner. The urinary nitrogen was also studied to determine whether the benzoic acid was sufficiently toxic to produce a disturbance in protein metabolism. The benzoic acid was given with breakfast, and the urine collected for the first four hours and kept separate from the remaining twenty hours' excretion. The hippuric acid was estimated on each of these samples by the method of Folin and Flanders.[7]

The output of hippuric acid in Case 1 was somewhat irregular, with a tendency to lag during the days of administration of benzoic acid. In the control patient most of the benzoic acid was excreted as hippuric acid in the first four hours following administration. Quick[8] states that hippuric acid is excreted at the rate of 1.4 to 1.82 grams per hour in man. The results obtained in our study suggest there was a disturbance in the availability of glycine in the patient with myasthenia gravis. Even though we attempted to deplete the available stores of glycine by giving this patient a diet

low in glycine for twelve days before administration of benzoic acid there evidently was sufficient glycine to handle the added benzoic acid, though the glycine was not readily available. Whether this is due to a disturbance in the body stores or to ability to synthesize glycine one cannot say.

Clinically, the administration of 3.3 grams of benzoic acid in Case 1 produced a very alarming effect on the patient. She became very much weaker. On the other hand, 3.3 grams of benzoic acid fed to normal persons caused no apparent disturbance. In Case 1 the average daily output of nitrogen during the five days' administration of benzoic acid was 6.66 grams. The subsequent five days' average output was 9.14 grams of nitrogen, but during this time 2.8 grams extra nitrogen were administered daily in the form of gelatine. If this added nitrogen is deducted from the latter five-day average very little difference is observed in the nitrogen output. One can conclude, therefore, that slight upset in the nitrogen metabolism was produced by the benzoic acid. It is interesting to note that patient 6, who has a good digestion of gelatine, is not benefited by the administration of glycine.

Two patients were given 45 grams gelatine daily for periods of two weeks in one case and three weeks in the other. Neither of these improved while receiving gelatine. With a view to replacing the possible lack of the enzyme which would liberate glycine one patient (Case 5) was given raw pancreas plus gelatine, but he became nauseated and his symptoms increased in severity. Later, raw pancreas, glycine and ephedrine were administered, but the weakness kept increasing. Within two days of the removal of raw pancreas from the diet the patient's condition improved greatly. It is probable that the pancreas, through its nauseating effect, produced the increase in the myasthenic symptoms. One wonders if in some cases the disease is not another deficiency disease.

SUMMARY AND CONCLUSIONS

1. All but one patient suffering from myasthenia gravis showed a diminished digestion of gelatine *in vitro* with the juice obtained from the duodenum.

2. One patient who received no benefit from glycine showed good digestion of gelatine.

3. Gelatine administration was of little value in patients suffering from myasthenia gravis.

4. The excretion of hippuric acid after the administration of benzoic acid was slower than normal.

5. Myasthenia gravis is probably a symptom-complex which has a multiple etiology. This is suggested by the variations in the response to different therapeutic agents and the digestive activity of the juice obtained from the duodenum.

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MORTALITY FROM APPENDICITIS.—H. Jensenius has analyzed the 1,933 cases of acute appendicitis admitted to a Danish hospital during the past ten years. Of the 1,592 patients who came to operation as many as 109 died—a mortality of 6.85 per cent. Compared with American and other statistics, with mortalities ranging from 2 to 5 per cent, this result is disquieting, and the author has indulged in some heart-searching. His comparatively high mortality may, however, reflect to a certain extent differences in classification; all his cases were acute, they all came to operation, all the appendices were subjected to a histological examination, and when this showed no sign of acute inflammation the case was not included in the analysis. There were 105 such cases without a single death among them. Of the 109 deaths more than half (fifty-seven) were due to general peritonitis; and in altogether seventy-two cases the cause of death issued directly from the appendix. There were as many as sixteen deaths from pulmonary embolism—a mortality of about 1 per cent from this cause alone. The ages of these 16 patients, 10 of whom were men, ranged from 39 to 65. With regard to the therapeutic lessons to be extracted from his analysis the author ventures the opinion that when a suppurative peritonitis is localized and in the early stage of abscess formation conservative treatment with opium is preferable to an immediate operation. But it is difficult to define such a condition clinically. When this can be done and conservative treatment is instituted, it seems probable that the mortality will be reduced.—*Ugeskr. Laeg.*, Nov. 5, 1936, p. 1085. Abs. in *Brit. M. J.*

GLAUCOMA, PRESSURE AND INFECTION

BY RICHARD KERRY

Montreal

IF a hypodermic needle be passed into the vitreous chamber of an eye and the pressure be raised there is no change in the depth of the anterior chamber, so that a shallow chamber cannot be due to hypertension. This experiment has recently been repeated several times by Dr. Stuart Ramsey, of Montreal, and no change in the depth of the anterior chamber could be noted. Paracentesis shows that there is no material obstruction to the passage of fluid between the chambers, and as the intake of the anterior chamber is so very much greater than the outflow the shallow chamber cannot be due to loss of fluid, and can only be caused by the iris being pushed forward by the swollen ciliary body. Inflammation is always accompanied by infiltration by leucocytes, and when the ciliary body is affected enlargement must follow. A glance at a magnified section of the anterior chamber will show that circulation through the canal of Schlemm will not be affected until after the anterior chamber has been largely obliterated, so that engorgement of the ciliary body must be the primary cause of hypertension and the effects of interference with circulation only secondary.

The conditions postulated above lead to two contradictory conclusions. The argument that as the intake of the anterior chamber is greater than the outlet the shallow chamber cannot be due to loss of fluid appears to be quite sound. On the other hand abundant evidence has accumulated since Priestly Smith's early work was published to show that acute glaucoma is associated with cyclitis. Swelling of the ciliary body cannot take place without something being displaced from the eye and that something can only be aqueous humour. Hence we arrive at an impasse. The first contention is true only if the secretion of aqueous humour is normal or increased. Should there be lessened secretion, with increased outflow, due to hypertension, the chamber will be depleted. The assumption of lessened secretion is not purely theoretical. During acute inflammation the flow of fluid from a focus is not sufficient to reduce pressure and under similar conditions in the ciliary body the secretion from it must be greatly diminished. In addition Niesmanoff's beautifully systematic work shows, that as tension rises the secretion of aqueous humour lessens progressively until at a pressure of about forty-eight millimetres of mercury it ceases.

The time-table for acute glaucoma is, uveitis, causing hypertension, lessened secretion of aqueous humour, and increased outflow; later interference with the flow through the canal of Schlemm, increased pressure rendering the tissue over it less pervious, and from viscosity of inflammatory fluid containing both colloid and debris; lastly, blocking of the canal by the iris. It is thus fairly evident that glaucoma is not due to blocking of the canal of Schlemm, and we are no longer confronted by an apparent contradiction of a blocked canal and a shallow chamber.

Some years ago the writer reported the results of the bacteriological examination of 8 cases of glaucoma. In 7 of these a growth of staphylococci was found on tissue removed from within the eye at operation; in the other case the tissue was accidentally brought in contact with a strong antiseptic and there was no growth. We were disposed to regard most of these cultures as being due to an attenuated germ which was probably anaerobic, as the growth was so slow. If it can be shown that staphylococcus infection is the cause of glaucoma the extensive enlargement of the ciliary body can be readily understood, and the reason why hypertension is so much greater in glaucoma than in any other form of cyclitis becomes apparent.

Evidence of infection is particularly strong in posterior glaucoma. None of the reported cases are characterized by cyclitis or hypertension but by inflammation of the optic nerve, with selective absorption of its fibres. There is no resemblance between the flat regular cupping of uveal glaucoma and the excavation, showing great loss of substance but no sign of pressure, which characterizes the so-called chronic cases. The conditions present are such as can only be caused by local infection; the

inflammation has been shown in different cases to extend to a variable distance up the nerve, and the chronic course shows that its virulence can be comparatively mild.

It may seem unreasonable to claim that the same germ can cause both extensive swelling and absorption, but the rich vascularity of the ciliary body affords vigorous resistance and reaction, and it is even probable that germs which are too attenuated to attack the uvea may obtain lodgment in the poorly-nourished nerve tissue.

The practical conclusions arising from the foregoing facts are that the optic nerve may be involved in any case of glaucoma, and that no operation intended to relieve pressure can have much effect on extra-bulbar inflammation of the optic nerve. As the nerve is inaccessible, the only way of helping these hopeless posterior cases is to attack the apparent underlying cause, that is chronic sinusitis. The problem thus becomes one of prevention rather than of cure, and the rhinologist rather than the oculist must decide whether operation or the use of the various rays, high frequency currents, anti-staphylococcus sera or toxoids, or any combination of these, will be more helpful in eradicating the disease. It cannot be too often pointed out that chronic sinusitis is usually ignored, and that even after nasal operation residual infection and reinfection are often neglected, with disastrous results.

In cases in which progress is slow or while waiting for more active measures to be instituted, filling the naso-pharynx twice daily with a mild antiseptic and retaining it for thirty seconds or longer is very helpful. If after the fluid has run out, the nose be blown freely without closing it, to remove mucus, and the treatment repeated, it will act more effectively on the cleansed mucous membrane. The use of iodine in oil, to stimulate leucocytosis and to raise resistance, is also indicated. If used two or three times a week remarkable results are often obtained.

The treatment outlined above may usually be depended upon to abort incipient glaucoma, and is of great benefit in any case, but if relief of tension is urgent this must not be neglected, although even after operation the condition of the naso-pharynx should not be ignored.

THE TREATMENT OF HERNIA BY INJECTION*

BY F. B. BOWMAN, M.B., F.R.C.P.(C.)

Hamilton

THE attention being given to the so-called injection treatment of hernia in the literature of late has prompted the following observations. The writer's experience with this method has been rather limited, and includes only 20 cases, but the results have been satisfactory.

To enter into a discussion of the reparative process of wounds and the part played by fibrous tissue in their repair would be elementary, but their importance in the repair of certain pathological conditions, hernia, hæmorrhoids, varicose veins, hydrocele, etc., has only of late received the attention that it should, and this because of the popularity of ambulant methods in treatment, which obviate the necessity of hospitalization with loss of time and expense to the patient.

Billroth stated fifty years ago that if a solution could be found which would cause the proliferation of connective tissue as dense as fascia the surgery of hernia would be solved. Such eminent surgeons as Halstead and Bassini popularized surgery in the cure of hernia; but, even under the best of surgical attention, the results have been anything but satisfactory. Statistics of from 5 per cent to 20 per cent of recurrences following hernioplasty have been published, and if all cases had been followed for ten years the second figure would probably have been more nearly correct. Others have intimated that the operative treatment of direct hernia has been so unsatisfactory as to create a major surgical scandal. When one realizes the disability and economic loss in the United States alone, where there are at least 5,000,000 persons with hernia, 80 per cent of whom are wearing trusses which are improperly fitted,

* Read before a Staff Meeting of St. Joseph's Hospital, Hamilton, Ontario.

the question of hernia and its treatment is still worth investigation.

The injection treatment for hernia has been in use for one hundred years, the first recorded operator being Velpeau, who used it in 1835. It has been used by different operators since then, but because of the necrosis produced by the solutions in use in the early days it was discredited, although cure was frequently obtained. Since 1925 thousands of cases have been reported, and much animal experimental work has been done, using different solutions and studying connective-tissue proliferation. In hernioplasty the removal of the sac, replacement of the cord, and the suturing process, play secondary rôles; it is the formation of fibrous tissue which is necessary for cure. The surgical profession generally has condemned the method as being unscientific and dangerous. Such a conclusion, from the experience of the writer who has had few cases and of those who have treated thousands, would seem to be without any foundation in fact. At the hernia clinic in the Minneapolis General Hospital 1,000 patients have been treated without a death, and in general with excellent results.

Too much importance cannot be attached to the operator being familiar with the anatomy of the parts concerned. This knowledge is as essential as in hernioplasty; and familiarity and skill in the use of injection methods is also essential to success.

All patients who are to be treated by the injection method *must* have a properly fitted truss. It is not sufficient to send the patient to the local truss fitter; one should accompany him and see that the truss is satisfactory. Where the hernia is direct the truss pad should be placed just above the ramus over Hesselbach's triangle. In indirect hernias the pad should fit over the inguinal ring so as to hold the hernial contents within the abdomen. Most operators prefer a rat-tailed type of truss, but one must use judgment, for this type will certainly not fit all cases. A man with wide hips of the feminine type, with a fat pendulous abdomen, may need an elastic truss. Others may need a perineal band to hold it down, for it must be worn day and night, and even in the bath, so that a waterproof one is preferable. A truss should always be applied lying down, and, to be satisfactory, must be comfortable. It should be no more uncomfortable than a new well-fitted pair of shoes. Once injection treatment has begun, the hernia must be *constantly* reduced. A truss may fit at one time and a month later be useless if the patient loses weight, so that it should be examined frequently to be certain that it fits. Where one wishes to treat a large abdominal or incisional hernia a special pad and belt may be necessary, and time spent in having this properly made and fitted is certainly not wasted.

Many solutions have been proposed, and nearly all of them depend on herbal extracts and tinctures combined with phenol and alcohol. Distillates of tinctures of different herbs have been combined with tannic acid in different proportions and are sold under patented trade names. Experimental work and treatment of cases have shown that connective-tissue proliferation seems to be stimulated to a greater degree by vegetable extracts such as thuja, pinus Canadensis, and cinnamon. Whether the reaction they produce is due to the presence of gallic or tannic acids one is unable to say. Pina-Mestre, the Spanish authority on this subject, who reports the treatment of more than 10,000 patients, uses a complicated mixture of herbal tinctures. Mayer, who has reported a large number of treated cases with excellent results, uses the following solution.

MAYER'S SOLUTION

Zinc sulphate	1 dr.
Phenol crystals	6 dr.
Glycerin	4 fl. dr.
Aq. cinnamomi	1 fl. oz.
Fl. Ext. Pini Canadensis (dark)	5 fl. dr.
Distilled water	2 fl. oz.

Dissolve the zinc sulphate in the cinnamon water. Liquefy the phenol crystals by heating. Add glycerin. Shake thoroughly until mixed and allow to cool, then add the distilled water and finally the fl. ext. of Pinus Canadensis. Shake thoroughly and allow to stand for a week with frequent shaking, and then filter. Before injecting boil the fluid to be used.

This, the writer has found gives excellent results and in his hands has shown no untoward effects. It is used in amounts ranging from 0.5 to 1.5 c.c. Macdonald's solution I have used occasionally, but it causes more pain, although the reaction with it is very good. Its composition is as follows.

MACDONALD'S SOLUTION

Phenol	50 parts
Alcohol	25 parts
Lloyd's specific tincture of Thuja	25 parts

Allow to stand two days and filter.

As regards methods of treatment, many different solutions have been used by many surgeons with excellent results. It does not make so much difference what solution is used as long as a regular routine of *careful* injection is adhered to and the patient follows instructions.

Inguinal hernia.—Knowing that the internal ring is situated 1 cm. above a straight line drawn from the anterior superior spine to the pubic spine, treatment is begun here. The hair should be clipped and the usual surgical precautions for injections anywhere are used. The patient is placed in a semi-Trendelenberg position, that is, with the shoulders lowered so that the hernial contents will gravitate toward the abdomen. A small syringe, such as a tuberculin syringe, is used and a 3 inch 22-gauge needle. Mayer's solution is shaken, boiled and slightly cooled, and then drawn up into the syringe. The needle is plunged perpendicularly through the skin and fat until the fascia is reached and a sudden give is felt as it is pushed through this into the ring. When this point is reached, it is noticed that the point of the needle may be swung from side to side and around in a circle very easily. Now suction is applied in case the needle may have entered a blood vessel. If blood appears in the syringe the needle is withdrawn slightly and suction again applied and if no blood appears a small amount of solution is injected. Then the patient is asked whether he feels anything, pain in the scrotum, which would indicate spermatic cord injury, abdominal pain or stinging, which suggests peritoneal irritation, tingling or numbness on the inside or anterior surface of the thigh, which indicates nerve injury. Some operators recommend the use of novocaine before the injection to relieve any pain. I have found this unnecessary and unsafe. One patient whom I injected with novocaine collapsed when he attempted to use the leg on the side which had been injected. The anterior crural nerve had been injured, hence the resulting paralysis. It was several hours before he could walk, and several weeks before he had entirely recovered. An injection given too superficially may cause a slow healing slough, even although the hernia may be cured. The injection is given twice weekly, gradually introducing the needle farther along the canal, and finally placing a few injections in Hesselbach's triangle so that an indirect hernia may not be turned into a direct one. Twenty injections may be required before a cure is effected, but frequently after three or four the ring may be closed. After the injection pressure is made over the area, the truss is replaced, and the patient allowed to get on his feet. Direct hernia usually requires more injections than indirect, and femoral hernia, fewer. I have had very striking results in two cases of hernia following laparotomy. One patient had a hernia as large as an orange in the epigastrium following a cholecystectomy. A special pad and belt were made for her, and after some twenty-five injections the abdomen was flat, although there was a large indurated area over the hernial opening. One must be particularly careful in these cases to be sure that no intestine is adherent to the abdominal wall. An injection into the peritoneal cavity would certainly cause plastic peritonitis and perhaps obstruction.

There are certain contraindications to this method of treatment, namely: hernia associated with undescended testicle; hernias which are incarcerated or irreducible because of the possibility of strangulation; reducible hernias which cannot be controlled by a truss; sliding hernias; and the usual surgical contraindications.

CONCLUSIONS

1. There is no danger in the injection treatment of hernia if the operator is skilled in this method, and in suitable cases it is as satisfactory as hernioplasty.

2. Recurrences are no more frequent than after operation, and if the hernia does recur, even in a mild degree, the patient may be reinjected.

3. The cooperation of the patient is absolutely necessary in the fitting and wearing of a truss, and the fitting should be done by the surgeon treating him.

4. The economic side of this method of treatment is important, and should be of interest to employers of labour and Workmen's Compensation Boards.

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SOME THOUGHTS ON ACUTE OSTEOMYELITIS OF THE LONG BONES*

BY R. B. DEANE, M.D., F.A.C.S.

Calgary

THESE remarks on acute osteomyelitis are intended for the man with a general practice, who sees such a case perhaps once in several years, and not for the surgical specialist, to whom what I have to say is of course mere A.B.C. The two most destructive conditions with which I have to deal are osteomyelitis and anterior poliomyelitis, and the amount of crippledom from these two sources is appalling, to say nothing of the occasional incidental loss of life, but earlier diagnosis would greatly help to reduce disability from osteomyelitis, and to this end these thoughts are offered for your consideration.

An important point is that the diagnosis can be made at the bedside, irrespective of the laboratory. In availing ourselves of laboratory facilities it behooves us not to neglect the information afforded by those simply applied clinical aids — the senses. In any event an x-ray in acute osteomyelitis is of no value, a point I particularly wish to emphasize, as there may be some who think that because the x-ray does not show anything there is no trouble in the bone, which is of course fallacious. On the other hand, a leucocytosis of 20,000 in an acute febrile condition, accompanying bone pain, almost certainly denotes pus; so a white count, if it can be readily obtained, is confirmative, but do not wait for it if the physical signs point to acute bone inflammation. Time presses and every hour sees an extension of the process. One of the most severe cases I have seen did not go beyond 13,000 white cells.

We must disabuse our minds of the notion that acute osteomyelitis is always accompanied by high temperature, excruciating pain, and marked constitutional disturbance passing into coma. There is such a picture, but that is the rare fulminating type. Be careful not to give morphine or other analgesics until your examination is made and the treatment settled, for the same reason that you would not in acute conditions in the abdomen: one cannot elicit the true state of affairs from a narcotized bone.

We will take the case of a child with an acute painful swelling of a knee region, accompanied by fever, which has followed a trauma. The occurrence of a trauma in such a case would make one suspicious, and, in passing, I wish to stress the fact that the trauma is generally quite slight; a fall is the common injury. A question or two will often draw out the history of the presence or recent presence of some cutaneous pus focus, which also may seem a quite inconsequential affair: *e.g.*, a stye, pimple, infected mosquito bite, or what not. We note that both temperature and pulse are somewhat raised. The temperature may not be above 101 to 102° F. Such are the ordinary common cases. Pain is always a prominent symptom. Indeed, I firmly believe that monarticular pain in a child with fever should cause the surgeon to think first and foremost of osteomyelitis. The pain is fairly localized to the bone involved, but there is some radiation. This pain is not excruciating in the ordinary type of osteomyelitis. The limb is held semi-flexed and is evidently very painful. In the case of this knee we are considering it is difficult sometimes to say from the history alone whether the lesion is above or below the joint. The moderate swelling is not red, which is noteworthy, and the affected part is distinctly hot to the feel. Now comes palpation, *the all important part of the examination,* which clinches the diagnosis and should make the surgeon confident of his treatment. Do not try to handle the limb; leave it lying as you find it, in the most comfortable position; otherwise you cause undue pain. Then with the tip of one finger, for convenience, the index finger, start about half way up the thigh on the inner side, if possible, and make gentle but firm localized pressure on the bone. The pressure points are made about one-half inch apart, passing down the femur in a vertical direction to its lower end. Be sure to keep up the pres-

* Read at the Annual Meeting of the Canadian Medical Association—Alberta Division, September 16, 1936.

sure for a few seconds at each spot and if the patient does not give the pain response in the femur, start on the tibia, beginning about the middle of the bone and work upwards towards the knee. It is the *pain response* to your localized pressure which gives you the certain clue to the site of the infection, as you will find that ordinarily the child will cry out bitterly. In any event, when once you have elicited the tender spot, do not keep repeating the test. In detecting these pressure points it is best to sit by the bedside quietly and take plenty of time.

Having found out where the trouble is, now is the time to give the hypodermic, if needed, and proceed at once to exploration. Acute osteomyelitis begins in the metaphysis which, as you know, is the narrow growing margin of bone on the diaphyseal side of the conjugal cartilage. Of the factors which determine the localization of the microorganisms in the metaphysis, such as the latter's peculiar vascularization and the effect of trauma, modern work indicates that lack of active phagocytosis in the metaphysis is of even greater import. The bone medulla is incomparably richer in phagocytic elements than the metaphysis; were it not, diaphysectomy would be required much oftener than it is. Acute osteomyelitis is much commoner at the growing end of a bone; *e.g.*, the upper end of the tibia, the lower end of the femur, the upper end of the humerus, and so on.

As regards treatment, one of the greatest surgical clinicians of our time, the late J. B. Murphy, used to say that a gimlet hole is sufficient to evacuate the pus, but this is probably an understatement, unless the disease is seen a very few hours from its inception. The late Clarence Starr advocated making several drill holes in the metaphysis. To ensure the best visibility and to save blood a tourniquet should be used. Soft parts and periosteum are incised to the bone. Do not, however, separate the periosteum from the bone to a greater extent than necessary. I use a good-sized drill (9/64") to explore the bone, and bore a number of holes over the tender area in a vertical direction, making sufficient holes to cover the pus area. Then with an osteotome join these holes together, being careful to remove all overhanging edges of bone, otherwise there will be difficulty in healing. ***On no account whatever use a curette to scrape the endosteum,*** which plays so important a part in the viability of the deep layers of the bone. Wiping out the cavity with a piece of gauze is amply sufficient. Drainage is the main thing. Another point is that should the pus lie under the periosteum you must drill on into the marrow and open up the bone as above. A subperiosteal abscess simply means that the pus from the marrow has escaped along the Haversian canals to the surface. In a doubtful case, do not hesitate to explore by drilling two or three good-sized holes in the bone, as no harm results if pus is not found, and do not forget that very rarely pus will appear two or three days later in such a case.

For the last ten years I have been using a 10 per cent solution of mercurochrome as a dressing, as I am confident that this drug lessens discharge, probably by an *intra vitam* staining of the microorganisms, and also favours kindly healing. It is necessary, however, to cover the dressing with a small piece of rubber tissue, to ensure capillary attraction. Otherwise the gauze would dry and act as a plug. Of course one uses a drainage tube where such seems best. I think too that a splint is an essential part of the treatment.

Occasionally, more than one bone is affected, *e.g.*, the humerus and tibia, or both tibiæ. The upper end of both femora with joint involvement is not so very rare, and is a very serious condition. These are difficult cases and require very prompt relief of tension by drainage. Otherwise the femoral head is likely to dislocate, which is nature's attempt to save the upper epiphysis from destruction. If dislocation does not occur the epiphysis is probably sacrificed, unless drainage is done very early. In multiple osteomyelitis the two affected bones do not progress with equal pace. Probably the disease does not start simultaneously in both, so that it may be necessary the day following the opening up of one bone to open another. For diagnosis:— bone pain and physical signs as before.

The need for early diagnosis was also recognized long ago by J. B. Murphy, who used to insist that the bone damage was done in the first twenty-four hours, but there is no doubt that, untreated, the damage increases progressively with the passing of the days, due, I believe, to a pressure necrosis, till at last one comes to the dead diaphysis, which remains a blanched and silent witness to a lost opportunity.

Recurrence following acute osteomyelitis is so common that, my impression is, that this is largely owing to delay in making a prompt diagnosis, which thereby allows the micro-organisms to "dig in" and become entrenched. Consequently, I have come to the dismal conclusion in a general way that, once osteomyelitis, always osteomyelitis. I feel that other factors, such as microbic virulence and poor resistance of the patient, play but a minor part in the recurrence. One has several times had to deal with cases of osteomyelitis that had remained healed for many years, from the time of the original attack in childhood, and then become acute, to say nothing of those subacute exacerbations which break out from time to time during the entire life of the patient. If the initial attack has run some weeks before treatment is instituted, convalescence will probably go over months, if not years, and then, after healing is complete, recurrence is the rule. I have not the least doubt that late diagnosis is the common cause of this feature of the disease, and in pointing the error I have tried to avoid exaggerating the truth. How tenacious of life is the word "rheumatism", which has been the chief setback throughout the years in the treatment of acute osteomyelitis! An acute bone abscess requires a much more urgent application of the ancient rule: *Ubi pus, ubi evacuo* than an abscess of the soft parts, but the impostor "rheumatism" creeps in and deceives the doctor, by giving him a false sense of security, which subsequent events are destined to shatter. Finally, if in our minds we could learn to associate bone pain and localized tenderness with osteomyelitis and forget all about "rheumatism" an enormous amount of crippledom and hospitalization would be saved.

ENVIRONMENT AND THE NEUROSES*

By RUTH MACLACHLAN FRANKS, M.A., M.B.

Toronto

THE subject of this paper is the study and treatment of those functional neuroses which, if untreated or not treated sufficiently early, may terminate in insanity.

Never has there been a time when so many persons have had to assume the burden of supporting others. People who put forth their best effort to maintain their own economic integrity during a period of social dysfunction are now faced with the problem of providing for those who are not capable of meeting life's difficulties. The neuroses may not be on the increase, but from the number of cases one sees each day, that is the conclusion that would naturally be arrived at. Whether or not the actual number is greater, the fact remains that the number of these unfortunates coming to the attention of the physician is multiplying rapidly. This is due to more extensive and better organized social service work, to the influence of periodic health examinations, to the recent period of stress and strain, and to a definite increase in incidence, if hospital statistics are consulted and physicians' opinions accepted in lieu of a general statistical survey.

HEREDITY

Heredity is a factor in the neuroses, but there is no uniformity of opinion as to the extent of the hereditary roll. In some cases the environmental integers are of primary importance, in others, the hereditary. When complete data are not available it would seem advisable to give heredity and environment equal consideration. It is readily observable from a genetical investigation that two healthy non-neurotic parents from a line of non-neurotic ancestors can have a child neurotic as a result of environmental influence. Conversely, the progeny of two neurotic parents, raised amidst healthy surroundings, may not develop pathological aberrations, but if left with the parents will show signs of the morbid state. Further, one can go so far as to say that two individuals neurotically constituted should not marry; neither should two persons mate when one is neurotic and the other prone to alcoholism, to drug addiction, to hysteria, to emotional instability, to

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schizoid personality, or to any form of mental disease or defect.

EPOCHS

The life-span of a normal individual or the so-called normal, carrying a hidden gene for the neuroses, can be divided into six epochs. These are the pre-school, the school, puberty and adolescent, pre-marital and marital, climacteric, and senile, and in each one there are one or two crucial points where psychic trauma may take place.

In the *pre-school period* there are two relationships to be established, the child-parent and the child-world, or social, state. Moreover, from three to five years the child has to learn more than during any other three-year interval in his life. Consequently, these early formative years are of primary significance in the determination of adult personality and character. The first obstacle in the way of a serene relationship between parent and child is the cry. For many years, the over-anxious parent was too concerned with the cry of the infant. The physician, recognizing the devastating effect on the mother, strongly advised ignoring the cry, and now the pendulum has swung to the other extreme. Children are permitted to cry for hours when some form of discomfort is the cause. The well-fed, well-nourished infant on a consistent, regular, daily program does not cry more than thirty to sixty minutes in twenty-four hours, and not investigating prolonged crying is harmful to the organism. The second discordant note is fear. One seldom gets a case of neurosis without a history of fears going back into childhood. Hundreds of parents use the fear-reaction as a means of control. Every day thousands of children are threatened with the bad dog, the policeman, the bogey-man, the doctor, bad medicine, etc., as a disciplinary measure, and thousands of adults are still carrying the scars of those early years and transferring them to their own children. Very few parents seem to know that when the sudden introduction of a new object produces fear in a child it should be removed and the child's attention distracted, later educating the child to the offender by a very gentle approach and kindly reassurance. The following case brings out these points very clearly.

A married woman of forty-five years, absolutely unable to make a social adjustment, came seeking advice. On inquiry, it was learned that as a child she had a great fear of feathers, and her whole childhood was controlled by the use of feathers. They blocked off forbidden areas of the house, stairs she must not climb, thresholds she must not cross, etc. For punishment, she would be placed underneath the kitchen table with each side bounded by feathers. There she would sit motionless from fear until released. Other fears naturally followed, and a teen-age marriage to a neurotic man completes a not uncommon story. Three children of this union have all completed their minority, and not one is self-supporting, on account of imaginary ailments.

With regard to the social relationship in this period the danger rests in introducing social contacts before the child is ready. From birth to two years the infant is self-sufficient. He does not require companionship and many happy, restful hours can be had by the child alone in the nursery. After two years play with other children is essential. Assure a child a definite time for uninterrupted play, avoid coercion and inexorable demands, make your commands simple and expect them to be obeyed, this is the advice to give to mothers, and they will then have the joy of seeing a healthy, happy child enter into the next part of life's journey.

In the *school period* the mingling with playmates continues, and to this is added the child-teacher relationship, a vulnerable point for some years. Again, the physician must work with the teacher in guiding the pupil. Between the child and his playmates one watches for the building up of inferiority and superiority complexes, the former, the more frequent and more harmful to the individual. Secondly, deviation from the average in any direction must receive thorough investigation and not be accepted as mental defect, genius, or some peculiar manifestation. In 90 per cent of the cases adjustments can be made which will prevent permanent injury. The teacher must be advised to inquire into day-dreaming, dull headaches, frequent micturition, seclusiveness, restlessness, etc. Just recently a case was brought to our attention.

The mother of a boy of eight years, in a supposedly very good boys' school, was advised that her son was suffering from a kidney condition because he asked to leave the room frequently. On inquiry, it was discovered that the boy had superior intelligence, and was not interested in the junior teaching that he was receiving. A transfer to a higher grade quickly cured the serious kidney condition, but not before it had given the parents considerable concern.

Puberty and adolescence is reached with considerable apprehension. Lack of parental instruction and the street urchin are responsible.

It is a reflection on our educational system that the discussion of sex with children is so embarrassing to parents. It should begin when first questions are asked in the pre-school period and gradually elaborated as occasion arises until the teen-age boy or girl is thoroughly equipped to meet this period—a very simple way to avoid mental damage in minors.

In the *pre-marital and marital states* the same lack of physiological knowledge leaves a loophole where fear, secrecy, and shame creep in to irreparably tarnish the best moments of life. One cannot over-emphasize these various danger points. Never has a history of a neurotic patient been taken without each one standing out in sharp relief to many minor, dependent, secondary factors.

One step further, *the climacteric,* another epoch surrounded by mystery. The menopause, like teething, is blamed for every symptom listed in almanac or medical textbook. Many women fully expect to become mentally ill during this period, and spend days in fear and dread of its onset. They are so obsessed with the idea that an uneventful cessation makes them even more alarmed. A recent patient told me that her glands could not be functioning properly because she felt so well and energetic since the climacteric, some three years before. The very fact of physical well-being meant endocrine dysfunction to this woman because she expected otherwise. Without medical help and reassurance this case would be diagnosed as chronic neurasthenia in ten years. It is therefore wise for the doctor to anticipate these problems when a patient of forty-five to fifty years of age is interviewed.

The *period of senility* is rarely accompanied by neurotic manifestations unless the patient is suffering from senile psychosis or has formerly been of a neurotic constitution. In any case it is too late to prevent the advancement of the disease, and the physician is left with palliative procedure.

DIAGNOSIS

The first step in treatment of the neuroses is diagnosis. No more grave error can exist than to overlook an obscure organic disease in favour of a diagnosis of neurosis. Such a conclusion should only be reached after a careful and thorough investigation of each system. This always means more than one visit, particularly as it is exceedingly difficult to get an accurate history owing to the variation in the patient's statements from day to day. Outstanding among the protean manifestations of the neuroses are mental and physical fatigability, hysteria, simple depression, and, frequently, dysmenorrhœa. If the disease has taken a hypochondriacal form the symptoms are referred to one or more organs and are changeable with suggestion. The viscera affected are always those familiar to the laity. One seldom has a patient referring pain to the pancreas or parathyroids. The neurasthenic has no interest apart from himself, does not exert himself, makes no attempt to share his life with others, is asocial and absolutely dependent. On arriving at a diagnosis always take into consideration the racial origin of the individual. The emotional reaction to the same situation in the Aryan and non-Aryan varies tremendously, and even in the Aryan group one must place different values on similar responses. Finally, as previously stated, full credit must be given to hereditary and environmental items.

Pause a moment to compare the repetitive phrases taken from a case-study of depressed, neurotic patients at the Toronto Psychiatric Hospital, with a quotation from the literature regarding a normal, healthy woman who suffers from a reactive depression due to a sudden tragedy in her life, the loss of husband and child.

Phrases: "I am ruined; I will never get better; my brain is gone; my body is diseased; something terrible is going to happen; I am dying; my heart is weak; I don't care; it doesn't matter; nothing matters; all is over; all is lost."

Quotation: "Time and Tide." 9.2.35.

"I remembered how I went about my work weeping, not knowing that I wept . . . . I could not find comfort. It was not that ropes were not lowered into that dreadful shaft to save me; there were friends, work, travel, even success; I did not lie supine at the bottom; but the ropes broke the minute I pulled on them to clamber out. For the first time in my life I did not escape from what I detested by the swift action of my will or some glorious, magical adventure. I had not guessed that I could ever be cornered, but I learnt that I was no exception; . . . . there were other phases when light went from day and dignity from thought; when the ache of what might have been put the stifling taste of ashes in all the real boons that life brought me. But these things, too, drew to a finish, though simply by the usual process of healing—slow, tedious and uninspired. Once more there was no sudden deliverance; nor did I prove to be a firm and magnificent conqueror of many kinds of despair." Finally recovery. "I knew that I had fashioned for myself, out of unspeakable, ignoble and necessary pain, a mind that faced reality, a road that was direct, and a spirit that could remain alone."

Throughout this whole passage one can see the effort extended to maintain balance and the endeavour to conquer. What a contrast with the morbid, hopeless pessimism of the phrases given. Two depressions, but how different the treatment from the medical viewpoint.

TREATMENT

The patient must be seen once or twice a week over a period of months. After rapport is established a careful individual analysis is made. Gradually the patient is brought back into contact with his surroundings, and in the meantime the physician has made sure, through communication with others, that his milieu will meet the requirements of this individual who is groping his way back to reality. In addition, other therapeutic measures must be employed. A well-balanced diet, emphasis being placed on all the vitamins, particularly B, as all these patients tend to be anæmic, is essential. The sluggish alimentary tract has to be rectified and free elimination encouraged. A tonic consisting of nux vomica and bromide is most beneficial. At least ten hours' sleep in twenty-four is necessary, accompanied by one hour's rest in the middle of the day at the beginning of the treatment. Lastly, outside interests and occupation must be introduced. This requires great patience, skill, and persistence on the part of the medical advisor. A detailed daily program should be written out for the patient. Throughout the country some of the larger centres have occupational therapists who can be appealed to for assistance. In Toronto, the Occupational Therapy Curative Workshop is invaluable in the treatment of these patients. Here the neurotic patient can find interesting employment in happy, cheerful surroundings and the tactful guidance of well-trained aides. It is not long before he acquires the necessary sense of security.

PROGNOSIS

The onset of definite neurotic symptoms under twenty-one years of age can be looked upon as grave. The majority of these patients develop one of the major psychoses in later life. Episodes occurring between twenty-five and thirty years, and between forty-five and fifty, are usually amenable to treatment. Occurrences at other age levels yield to treatment too, sometimes manifesting very gratifying results, but they require a longer course of treatment and a greater expenditure of effort on the part of the physician and those in attendance. Each successive let-down is greater in severity and longer in duration than its predecessor. Early treatment of the neuroses does decrease the incidence of the psychoses.

PREVENTION

Prevention begins with the awareness of the danger points as outlined in the various epochs. Not only the parent but the physician too must be taught to be continually conscious of good mental and physical hygiene. Educational centres and mental health clinics throughout the country prevent and alleviate many unnecessary painful situations. Social organizations and groups must be part of everyone's life in order to build up outside interests and maintain good social adjustment. The medical man has to be more careful in his counsel to patients, and particularly to parents of children. This is so often true in cases of head-injury when unnecessary restriction is placed upon the child's activities after recovery. The child is constantly reminded of his injury and establishes many other associated ailments. If his youthful exuberance breaks bounds the parent is terrified that death will intervene. Similarly, it is not uncommon to have a child's play life very limited because of a heart condition that does not exist. Chronic invalidism due to an error in judgment of someone who should know better is regrettable. Hundreds of children have been reared by over-anxious parents to believe that they are delicate or nervous. In fact, it is not uncommon for the child to take a certain amount of pride in his disability, and seek attention in his group by describing in minute detail his illness. One could hardly expect such a child to grow into a healthy-minded adult. Psychotherapy must be the keynote in the treatment of every case, and work the safeguard against the neuroses. To quote from Sacha Guitry: "I am convinced today that the man who does not love his work, who works without joy and for the sole purpose of earning a livelihood, is the most miserable of men." If attenuating circumstances necessitate a man doing work which does not give him pleasure, then it is absolutely essential that a

hobby outside of working hours must exist, to provide the joy that Guitry describes. I have yet to see a happily occupied person suffering from a neurosis.

SUMMARY

The neuroses have been reviewed from the hereditary and environmental viewpoints. Special emphasis has been placed on critical periods from birth to old age. Good mental hygiene in early life is introduced as a major factor in prevention. Once the disease has developed, psychotherapy, good physical health, and occupation will hinder its progress, and even enable a person to adapt himself again to his environment. Etiological factors have been traced to thoughtless attitudes on the part of parent, teacher and physician.

TWO CASES OF EARLY SECONDARY ABDOMINAL PREGNANCY WITH MASSIVE INTRA-ABDOMINAL HÆMORRHAGES

BY J. O. BAKER

Edmonton

ABDOMINAL pregnancy is such an interesting subject, presenting so many varying features, that to have two unusual cases within the space of two weeks was considered sufficiently noteworthy to report.

The two cases were of the early type, and both were complicated by immense abdominal hæmorrhages. Cornell's and Lash's[1] collective review of the subject includes 236 cases, but even in this article no mention can be found of similar severe abdominal hæmorrhage in early cases. Falk and Rosenbloom[2] reviewed 313 cases of extrauterine pregnancy occurring in 17 years at the Harlem Hospital, New York, among which there were 5 secondary abdominal pregnancies, but they apparently had no case of early abdominal pregnancy with massive hæmorrhage.

Secondary abdominal pregnancy, and most cases of abdominal pregnancy are such, is bound to become more rare, for with the early recognition of tubal pregnancy, aided by obstetrical tests such as the Ascheim-Zondek, etc., and the prompt application of surgery, the embyro has less chance to be discharged into the abdominal cavity through tubal rupture or tubo-abdominal abortion. If it happened to be discharged very young, with the damage that has likely taken place in its exit from the tube, it would not stand much chance of continuing its development. It would be more likely to start up a peritoneal reaction and be absorbed or encapsulated. It can be imagined that it might continue if it had active trophoblasts and a suitable soil such as an endometriomatous or a decidual area. Older embryos escaping with the amniotic sac intact, and the placenta continuing to occupy its location with maternal connection fairly well maintained, stand a better chance. The placenta could possibly develop and grow over and attach itself to other areas. Therefore these cases usually have their attachments low down, as did these two cases about to be described.

CASE 1

A young married primipara, aged 27, of low and unstable mentality, came to me while the pregnancy was probably still tubal, complaining of pain low in the left side of the abdomen. Her last menstrual period was August 7, 1935. She complained of some "spotting", but refused proper examination. She left the office with the idea that the purpose of the vaginal examination was to abort her. It was impossible to reason with her. A few days later one of the group was called to the house where she was employed as a maid, and found her complaining of slight abdominal pain and some bleeding. She was admitted to the Royal Alexandra Hospital (October 13, 1935), but again absolutely refused to have a vaginal examination. At this time her hæmoglobin was 67 per cent; red blood cells 4,100,000; white blood cells 18,800; polymorphonuclears 96 per cent; lymphocytes 4 per cent. Her sedimentation test showed a drop of 12 in 60 minutes. The Ascheim-Zondek test was positive. Tubal pregnancy was suspected. She became extremely violent mentally. During this time she complained of some abdominal discomfort, had a slightly darkened vaginal discharge, but refused to have any further examinations or blood counts. She insisted on leaving the hospital on October 23rd. Following this she came into the office on several occasions reporting that she was feeling much better, but would not submit to any examination. In the early morning of November 7th one of the group was called to see her in her room. She had been flowing slightly for two days and complained of some sharp spasms of pain in the abdomen. A hypodermic of morphine was given her, and she was again admitted to the Royal Alexandra Hospital. Her white blood count was 12,000; polymorphonuclears 78, and lymphocytes 22. She still complained of some cramp-like pains in the abdomen. She vomited twice during the morning, and during the next few days was most difficult to attend, refusing examination or medication, but took her food regularly and did not seem to be suffering from much abdominal pain. There was very little vaginal discharge. On the morning of November 11th, she was most antagonistic,

but had vomited, was nauseated, and appeared to be having quite severe abdominal pain. Her temperature dropped to 97° F.; she looked extremely pale; and her blood pressure was 92/60. Her hæmoglobin was 48 per cent; red blood cells 3,150,000; white blood cells 21,350; polymorphonuclears 96; lymphocytes 4 per cent. She was persuaded to submit to a pelvic examination, which could not be done satisfactorily, but a sensation of bulging was obtained in the cul-de-sac and a mass felt in the left pelvis; also some fresh bleeding from the uterus was noted. She was immediately matched for a transfusion but refused to have it and was taken to the operating room. Under anæsthetic pelvic examination confirmed the diagnosis of abdominal hæmorrhage. A mass about 5 cm. in diameter was felt in the left fornix. The *operation report* was as follows.

"A mid-line incision was made. On opening the peritoneum a large quantity of blood, both old and fresh, was apparent. As much as possible of this was suctioned out, citrated, and used for immediate autohæmofusion. Abdominal pregnancy, with sac complete down behind the uterus; placenta attached to the left tube, ovary, broad ligament, and sigmoid. On examination to determine the point of hæmorrhage the sac was accidentally ruptured and a live fetus delivered. Some of the placenta which seemed loose was removed, and a large curved clamp was placed on a pedicle-like piece which was attached to the sigmoid and peritoneum; which piece it seemed unwise to attempt to remove. This was sutured over and the clamp removed, thus practically controlling the free hæmorrhage. There was still some oozing on the left broad ligament and cul-de-sac. Some packing gauze was put in, the end left protruding from the abdominal wound."

The patient had received 500 c.c. of her own blood by this time, but as we anticipated trouble in post-operative treatment, 300 c.c. of her husband's blood was given before the effect of the pre-operative sedatives wore off. As expected, she refused to consent to any post-operative treatment, and was most indignant about having been operated on. She did practically as she wished regarding taking the fluids and so forth. Her temperature was only elevated above 100° F. once. Removal of the packing was started on the second day. Some decidua was passed from the uterus on the third day. The patient otherwise made an uneventful recovery and left hospital on the 16th day.

The *pathological report* was as follows. — Fetus about three months, part of placenta and ovarian tissue showing placental attachment — intra-abdominal pregnancy. Tube not differentiated on resection.—M. E. Hall, Pathologist.

The patient has been seen several times since leaving the hospital and submitted to a pelvic examination about January 1st. The pelvis seemed surprisingly clear and the uterus about normal in size and freely movable.

CASE 2

The second case was referred on November 23, 1935, para-2; the last menstrual period was October 3rd. On October 9th she had been admitted to the Royal Alexandra Hospital to the service of the doctor who later referred her to me. She complained of pain in the lower right quadrant of several days' duration. She had had a similar attack, she thought, twelve years before. Urine examination was negative. As pain and tenderness had persisted a diagnosis of subacute appendicitis was made and operation decided on. The *operation notes* read.—"Right rectus incision, appendix free, full and slightly congested; long mesentery".

As the woman was known to be pregnant, her last menstrual period being August 3, 1935, and nothing abnormal was apparent, the surgeon did not pull up the tubes or investigate the lower pelvis.

Pathological report. — "Chronic productive and catarrhal appendicitis, with scars", M. E. Hall, Pathologist.

The patient recovered, with no marked abdominal pain. On October 18th she was flowing slightly. She was out of bed on October 20th, and discharged on October 23rd.

She was re-admitted on November 11th, the complaint being uterine hæmorrhage accompanied with backache which had developed a few days after leaving hospital. She was vomiting on admission, and her pulse was of a fair quality. She complained of some difficulty in breathing and required a hypodermic of morphine, grain 1/6, for pain. Apparently an impacted uterus was considered to be the disturbing factor and an attempt at replacement was made. As considerable pain resulted from this an anæsthetic was given and the uterus pushed up with some difficulty. An emesis of several ounces once or twice per day continued for several days and she complained of some occasional abdominal discomfort. On November 14th the nurse records.—"No emesis today". She was apparently better for a few days following this, although she seemed to have had indefinite pains, as recorded, in the bladder, epigastric, and lumbar regions, also some headache. Summarizing from the 18th on, when the indications were that something was developing, the points of interest were as follows.

November 18th.—Although having slept soundly all night she vomited ten ounces of undigested food; temperature 99.4° F., pulse 90.

November 20th.—She was crying with abdominal pain more severe in nature and had codeine, 1 grain, several times; only a fair day.

November 21st.—The condition of the previous day was apparently more exaggerated. Pain in the abdomen was more severe; frequent emesis; difficulty in breathing. She had codeine, 1 grain, three times.

November 22nd.—The patient seemed very listless, distended, and complained of pain over the abdomen; nauseated.

November 23rd.—The abdomen was distended and tender. My report on requested consultation was as follows.

"Apparently the abdomen is filling up with blood, dull in the flanks, etc. The patient looks exsanguinated. Pelvic examination reveals bulging fornices. Uterus movable, but cervix up behind symphysis; mass to left."

Diagnosis. — Hæmorrhage in abdominal cavity. Possibly abdominal pregnancy.

Red blood cells 2,550,000; hæmoglobin 40 per cent; white blood cells 10,200; polymorphonuclears 90, lymphocytes 20. Blood pressure 90, systolic.

The patient was immediately matched for transfusion, and was given 500 c.c. of citrated blood. As soon as possible under ether anæsthesia a laparotomy was performed.

Operation report.—Abdomen distended in all quarters, and, on opening fascia, the peritoneum bulged through, distended with blood. Blood was suctioned out of the abdomen for autohæmofusion. Over 1,500 c.c. were obtained. Aside from this many old clots were turned out. Abdominal pregnancy, left lower abdomen, attached to the cul-de-sac, left broad ligament and sigmoid and posterior surface of the uterus. On examining for the attachment of the sac, which had apparently ruptured, a live fetus, about sixteen weeks, was brought up and a portion of the placenta unfortunately detached. Four pieces of large packing gauze were immediately put down into the lower pelvis, the ends brought out through abdominal incision, hoping to control the bleeding from the placental site, which was very profuse, and the abdomen rapidly closed as patient's condition was now bad. Time of

operation: twenty-five minutes. During this time she was being autohæmotransfused, and this was continued until she had been given 500 c.c.

Her condition improved somewhat. Systolic blood pressure, which had been down to 65 during operation, went above 100; then oozing was noted around the packing protruding from the abdominal wound, and the patient's condition indicated she was bleeding again; blurring of vision, weak pulse, air-hunger. Her blood pressure would not register and she died four hours after operation. *Pathological report.*—"Fetus and placenta about three months", M. E. Hall, Pathologist.

It is interesting to note that both fetuses were located in the left lower abdomen, and to conjecture how much this had to do with the hæmorrhages. The bleeding in the first case certainly was not from examination or manipulation, and the second patient survived manipulation, etc., for several days.

Since the preparation of this article the June number of the *American Journal of Obstetrics and Gynæcology* has come to hand, and Reel and Lewis[3] of the Department of Gynæcology, Ohio State University, report 10 cases of secondary abdominal pregnancy occurring in their service in seven and one-half years. About 4 of these were early, in the first trimester of pregnancy or slightly later. Among these early cases they apparently had none with massive abdominal hæmorrhage. In their 10 cases 6 were located in the left lower abdomen, an indication of a possible tendency for secondary abdominal pregnancies to develop there.

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AN INTERESTING FAMILY HISTORY OF APPENDICITIS

BY G. G. LECKIE, M.D., L.M.C.C.

Lucky Lake, Sask.

THIS is an unusual history of appendicitis in one family.

The father's father died in 1885 at the age of 50, from "inflammation of the bowels", now known to have been appendicitis. The other coeval relations died in old age from unknown causes.

The father died in 1926, aged 52. He had had attacks of "indigestion" off and on for several years. In 1926 during an attack which was diagnosed as "indigestion" by a doctor, he returned home several miles in the country, became worse, and came into hospital with generalized peritonitis. A drainage operation only was done. Six days later he developed phlebitis in the left leg and died.

The mother, aged 58, has had frequent attacks of abdominal pain with vomiting. The pain is mostly in the lower right side but she has never consulted a doctor regarding it.

Daughter M. died in 1899, aged 15, after illness for several days. She died on her way to hospital. A post-mortem showed a ruptured appendix as the cause. Daughter C., aged 35, married, has had several attacks of abdominal pain which were twice diagnosed as appendicitis by different doctors — once this year. Daughter B., aged 33, married, has had a severe attack this year, diagnosed as appendicitis by her doctor. She refused operation. Daughter R. died at the age of 6 months, 1904, from "dysentery". Daughter B., aged 30, at nine years of age had a severe illness and was then said to be too sick to operate upon (she then lived fifty miles from a hospital). Ever since she has had pain in the side with "stomach trouble". At operation this year she had a short, thick and inflamed appendix (half sloughed away) with omental adhesions to pelvic wall; recovery. Son R., aged 27, has no history of abdominal complaints. Son J., aged 25, has had appendicectomy elsewhere in 1927. Daughter D, aged 22, in 1934 came into hospital with pelvic peritonitis. She had appendicectomy and drainage, and recovered. Son F., aged 20, had acute appendicitis this last September. Appendicectomy was done, with uncomplicated recovery. Daughter, aged 18, last year had a subacute attack of appendicitis which subsided. Daughter M., aged 18, has no history of abdominal complaints. Daughter A., aged 15, in 1933 came to hospital with acute appendicitis and peritonitis (localized). Appendicectomy with drainage was done, followed by recovery.

Questions repeatedly asked attending physician by family are: Is appendicitis hereditary? What can be done to prevent it? Should we all have an appendicectomy? What are the answers in the face of this history?

Case Reports

A DENTAL PROSTHESIS IN THE LEFT BRONCHUS: BRONCHOSCOPY: REMOVAL

BY J. N. ROY, M.D., F.A.C.S.

Professor in the University of Montreal, Montreal

Miss V.M., aged 27, came to Notre-Dame Hospital on February 21, 1936. She said that the preceding night, about 4.00 a.m., she was awakened by a choking feeling. After her breathing became less laborious she got up to take a little water, and then felt a sensation of malaise in the left side of the chest. Going back to bed, the patient did not sleep during the remainder of the night. She then noticed that the dental prosthesis she wore the preceding evening had disappeared. This piece, which had hardly been one year in use, had never been solid, and the patient was obliged to remove it off and on, especially at meal time. Occasionally she was compelled to apply a little cement in order to hold it fast to the abutment tooth. During the forenoon, she took her breakfast, but was unable to keep it down.

Upon examination of the mouth I discovered that the prosthesis had been made to replace the upper left central incisor. I observed a well defined inflammation of the mucous membrane in the rhino-pharynx. The larynx presented a slight degree of hyperæmia, but there was no erosion. I asked for a radiograph, and Dr. Laquerrière located the foreign body in the left bronchus. It was, however, impossible to discern whether the artificial tooth or its supporting metallic crown occupied the inferior position. We can nevertheless suppose that the tooth, being heavier than the rest of the piece, would be below (see Fig. 1).

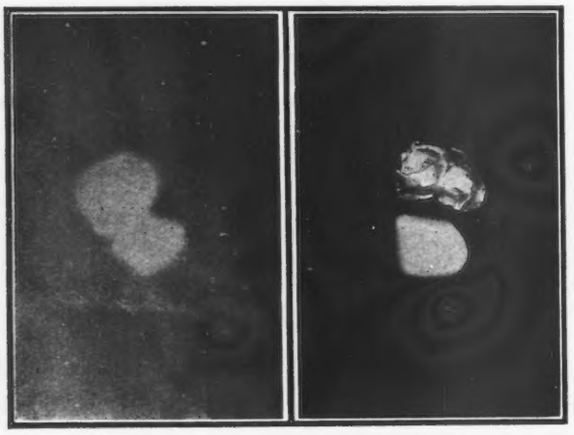

Fig. 1 **Fig. 2**

Fig. 1.—Radiographic aspect of the foreign body. **Fig. 2.**—Dental prosthesis, natural size.

Upon auscultation, Dr. J. Prévost observed that there existed a para-hilar congestive focus on a level with the middle third of the left lung. No sign of atelectasis existed. The right lung was normal. The patient was then admitted to the hospital, and the following morning her temperature was 101° F. Half an hour before bronchoscopic intervention a hypodermic injection of H.M.C. (hyoscine, morphine, cactoid) was given.

Operation.—Local anæsthesia of the pharynx and larynx with a 10 per cent solution of cocaine, by means of an atomizer; anæsthesia of the trachea with 3 per cent cocaine, with an intra-tracheal syringe. The patient being in a prone position, her head was forced back on the Haslinger's head rest. I used Chevalier Jackson's instrumentation. I passed a bronchoscope of 40 cm. in length, by 8 mm. in diameter into the left bronchus and immediately perceived the gold crown of the prosthesis. A flat-surfaced forceps was introduced, but slipped off the metal. I then had recourse to Tucker's forceps, and succeeded at once in removing the foreign body, which was located in the main bronchus about 4 cm. below the spur. The operation lasted six minutes. A second direct laryngoscopy showed that the vocal cords were undamaged. Post-operative sequelæ were normal. The temperature, the malaise in the chest, and the pulmonary congestion disappeared.

The prosthesis measured 17 mm. in length, by 11 mm. in its largest diameter. (See Fig. 2). The piece consisted of a porcelain tooth supported by an open-faced gold crown. This crown was broken at the labio-gingival border and presented cutting edges. The appliance weighed a little more than two grams.

Intrigued by the fact that the foreign body was lodged in the left bronchus, when from an anatomical point of view, it should rather have been located in the right one, I tried to find the cause. The patient was once again questioned and remarked that she was in the habit of sleeping on the left side. At the time of the accident, she declared that being awakened by a choking sensation, she leaned on the left elbow, and that she kept this position until the laryngeal spasm had disappeared. Being a heavy cigarette smoker, she presented a chronic pharyngo-laryngitis which causes violent coughing from time to time.

This report is interesting for two reasons: first because of the history of the case, and, secondly, because of the location of the dental piece. It is indeed rare that a person accepts a prosthetic appliance which she must remove now and then, and which she tries to restabilize by means of cement. The night of the accident the patient was, as always, perfectly sober, and it is more than probable that the cough, due to the irritation from tobacco, produced the aspiration of the prosthesis.

In so far as the foreign body in the left bronchus is concerned, we should have found it on the right side, rather than on the left, for the following anatomical reasons. The trachea of an adult presents on an average a diameter of 24 mm., the right bronchus a diameter of 23 mm., and the left one of 20 mm. only. Moreover, the spur of the bronchus is slightly inclined towards the left side. If the prosthesis localized itself on this left side we must admit the statement of the patient, that is to say, that, awakening with a choking sensation, she leaned on the left elbow and that she kept this position until the spasm

of the glottis had ended. At this moment, the dental piece, relatively heavy, having been aspirated, slid into the bronchus corresponding to the inclination of the thorax.

Although the radiograph did not show the precise position of the tooth it was logical to conclude that this porcelain would slide down first, due to its being the heavier portion of the appliance, and, due to its smooth surfaces, it would slip along into the left bronchus, carrying with it the metallic support.

In dealing with this foreign body, it was necessary to remove it without injuring, if possible, the vocal cords and the sub-glottis. Also, having introduced the bronchoscope to within the field of the dental piece, I realized that the gold crown with its sharp edges was indeed uppermost. A first attempt with the flat forceps was unsuccessful, due to its slipping off the metal, I then used the Tucker's forceps which allowed me to immediately remove the prosthesis. During this manipulation I took great care that this prosthesis would remain in direct contact with the bronchoscope, so as not to tear the tissue, especially at the region of the glottis. We know full well that a lesion of this part can have serious consequences, by producing an œdema, sometimes necessitating a tracheotomy. In this case it was necessary to exercise even more precaution due to the fact that the irregular and cutting metallic crown was directed upwards.

I do not think it necessary to elaborate further; I simply underline the fact that it is always very dangerous to wear a dental prosthesis which is not solidly fixed to the supporting teeth.

A CASE OF SO-CALLED POST-TRAUMATIC PSEUDO-PANCREATIC CYST

By William Oliver Stevenson, M.B., F.R.C.S.(E.), F.R.C.S.(C.)

Hamilton

S.G., male, aged 58, a farmer, was admitted to the Hamilton General Hospital on October 7, 1935.

History of illness.—On September 2nd, while removing a fallen branch from in front of his horses, they became frightened and bolted. He was trampled on by one horse on the upper part of the abdomen, the hoof sliding off the left lower ribs. He was able to get up and walk to the house, and, though somewhat distressed, returned and stabled the horse. An hour later, a doctor was sent for on account of the pain in the upper abdomen. The patient was put to bed with hot applications and sedatives and remained in bed for ten days. The pain shifted to the lower abdomen and he had considerable trouble with urination and constipation. On September 14th he was able to get up and remained up until September 18th, on which date the severe pain in the upper abdomen returned. This pain was constant and was referred to the front aspect of the left shoulder joint. His bowels continued to be constipated, and from that time until his admission to hospital he was troubled greatly with distension and gas and a slowly enlarging abdomen. He never vomited nor felt nauseated at any time.

Physical examination.—On admission, the patient looked ill and complained of general abdominal distress, the pain being dull and steady. No nausea or vomiting; no referred pain. His temperature was normal; pulse 110; and respirations 22. The abdomen was very distended, and no peristaltic movements could be seen or heard. The patient was given a high enema, with some relief, and a gastric lavage gave further relief. On the morning of October 9th; he had relapsed to the condition on admission. A further enema was able to bring away a little gas but gave no real relief. An intravenous injection of 1,000 c.c. of saline and glucose was given. Following this he was greatly distressed and complained of pain in the left shoulder. *Diagnosis.*—A tentative diagnosis of paralytic ileus was made, and the patient was taken to the operating room with the intention of performing an ileostomy.

Operation.—Under a local anæsthetic the abdomen was opened through a left rectus incision about the level of the umbilicus. About one pint of peritoneal fluid was aspirated. The small bowel was seen to be normal in size. Upon passing the index finger upwards in the abdomen it was met by a diaphragm which extended across the width of the abdomen and was tensely fluctuating. Pressure upon this made the patient complain of pain in his left shoulder. The condition was recognized at this point and the abdominal incision was enlarged one inch and a half upwards. The transverse colon was not seen. The lower abdomen was packed off. An anæmic area through the great omentum was pierced with dull-pointed forceps. Four quarts of milky fluid were aspirated, the terminal few ounces being a little blood-stained. A large tube was inserted. The edges of the cyst wall were sutured to the peritoneum. The abdomen was closed and patient returned to bed. There was further slight drainage from the wound, but the patient made an uneventful and complete recovery.

The pathological diagnosis of the fluid was that it was inflammatory exudate and contained red and white blood cells.

This case was one of so-called post-traumatic pseudo-pancreatic cyst which arises in the upper abdomen in the post-peritoneal space. The peritoneum on the posterior wall of the abdomen had been pushed forward and below the transverse meso-colon, to be covered by the great omentum. The wall of the cyst was therefore formed by the retro-peritoneal wall of the abdomen posteriorly, the peritoneum plus the four layers of peritoneum forming the great omentum anteriorly. The inflammatory fluid was an evidence of the reaction following an injury of the pancreas which was not sufficient to destroy that organ.

A CASE OF HYPERINSULINISM

BY GEORGE H. HAMLIN, M.D.

Austin, Man.

Miss M.H., aged 19.

Entrance complaints.—Frequency and nocturia for 4 years; swelling of the ankles and upper eyelids for 4 years; constipation for 4 years; repeated epistaxis since childhood; anorexia and nausea after meals for 2 years; four attacks of unconsciousness in 2 years.

Present illness.—The patient was a normal healthy girl in every respect until four years before. The first symptoms noted were frequency of urination during the day and a necessity to rise once or twice during the night. Constipation was first noticed about the same time. At times during the past four years she had noticed swelling of her ankles and upper eyelids when she got up in the morning. She stated that this cleared up after she had been up for a time. She had always had an excellent appetite, particularly for starchy foods, until about two years ago, but since that time her appetite had been becoming poorer, and now she did not want to eat because when she did she felt nauseated and occasionally vomited. She thought that she had lost about ten pounds in weight in the previous two months. During the past two years she had had four attacks of complete unconsciousness. These attacks came on without any warning, and as soon as they were over she could get up and proceed with her work as though nothing had happened. The first attack occurred two years ago and lasted for eight hours. During the next two months she had two more attacks which lasted for about two hours. The last attack occurred about two weeks before consultation and lasted for four hours. The first and the last attacks came on in the morning just as she was getting out of bed, while the other two came on in the middle of the afternoon. The time at which attacks of this nature occurred is of diagnostic importance.

Physical examination.—The patient was a well nourished girl of 19, and a physical examination made following the first attack was essentially negative. A blood sugar taken within an hour after the first attack was over gave a reading of 88 mg. and her blood urea was 20 mg. A definite diagnosis was not made at this time, and she was not seen again until the last attack occurred. The essential points on physical examination at this time follow. The patient was lying quietly on the bed and on casual examination appeared to be asleep but could not be roused. The skin was moist, and at first she was pale but after she had been unconscious for a time the skin became flushed. The pupils were widely dilated but showed a sluggish reaction to light. The respirations were shallow, and about 20 to the minute, and the pulse was 80 and of good quality. The blood pressure was 110/80. The abdominal reflexes were brisk, while the knee jerks were sluggish. A negative plantar response was observed. About fifteen minutes before she regained consciousness her condition changed from one of flaccidity to one of extreme restlessness, and she threw herself all over the bed. On regaining consciousness she felt well, except for marked hunger.

The problem here was first one of diagnosis, and as one could rule out the commoner causes of coma further investigation of the sugar metabolism was decided upon, in spite of the normal blood sugar reading following the first attack. She was given 100 g. of glucose and a fasting and one and two hour specimens of blood were taken. At this point the patient became very weak and felt faint and nauseated so that no further blood was taken. The results of the tolerance test were: fasting, 71 mg.; 1 hr., 65 mg.; 2 hr., 44 mg.

With these findings one is justified I think in making a diagnosis of hyperinsulinism. In view of the fact that in many of these cases the disease is due to a tumour of the pancreas, which is often benign, an exploratory laparotomy was considered but was refused by the patient. The treatment followed in this case was to outline a low carbohydrate diet with frequent meals, and in the event of any further attacks to give intravenous glucose and adrenalin.

RUPTURE OF THE SPLEEN

BY J. P. BONFIELD, M.D., M.R.C.S. (LOND.)

Ottawa

The patient, a male, aged 27, while riding in an automobile, was struck by a streetcar and a short time after was admitted to the accident ward in the hospital. He was quite bright and talkative while being examined, and was inclined to laugh at his injuries. He had a left "black eye", and a few superficial cuts about the face. There was some pallor, but nothing marked, and he assured us this was his natural colour. His pulse rate was around 88 or 90, which we assumed to be due to excitement. The following day, after an x-ray examination of his head, which revealed no fracture, he appeared so well that he was allowed to return to his home in the city. The next morning he came downstairs feeling perfectly well, when suddenly he was seized with great abdominal pain and collapsed on the floor. I saw him a short time afterwards. He was completely collapsed. He, obviously, had suffered a great internal hæmorrhage, and his pain seemed to be in the left side below the ribs. A diagnosis of ruptured spleen was made. The patient was re-admitted to the hospital immediately, and a transfusion of 700 c.c. of blood given. This was given not only to compensate for the blood lost but also for its hæmostatic effect. The catastrophic condition of the patient immediately changed to one of hopefulness, and he was left alone until the following morning.

An ample incision, about eight inches long, was made in the left rectus sheath; a small incision in these cases will prove most embarrassing for the operator. The great omentum appeared stained with blood, and as soon as this was disturbed dark recent blood began to pour out of the wound (about 1,500 c.c.). The spleen was sought and appeared to be enormously enlarged. It was delivered with considerable difficulty, and was about three and one-half times its normal size, the dark bluish tint denoting recent hæmorrhage into its substance. In delivering it into the wound, gushes of blood escaped from a small wound on the gastric aspect about the size of a fifty cent piece. The pedicle was transfixed, care being taken to avoid the tail of the pancreas, which is in such close proximity to the lower pole. The toilet of the peritoneum was carried out and the abdomen closed. The patient was again transfused while on the table. About 700 c.c. of blood were given.

The progress after this was uneventful. His temperature never rose beyond 101° F., and was normal on the fifth day. His pulse rate ranged from 120 to 90 for the first six days, and on the 23rd day had never dropped below 88. On the eleventh day a red cell count showed that he had 3,000,000 red cells and 32,000 white cells. On the fourteenth day his white cell count had risen to 43,000, and on the 23rd day had dropped back to 12,000. The enormous number of white cells in the blood stream would probably be doing police duty while the reticulo-

endothelial system as a whole was regaining its balance after the loss of such an important member. The patient was allowed out of bed on the seventeenth day. Thrombosis of the portal system is to be feared in these patients, and we deem it wise to give them a fairly prolonged stay in bed.

The patient was discharged from hospital on the 23rd day, apparently none the worse for his experience, and in quite a happy mood, which seems to contradict the older writers, who state that, as a rule, very happy people and great laughers possess great spleens.

The diagnosis in this case was at first masked, because the hæmorrhage had taken place into the splenic substance, which allows great stretching because of its fibro-elastic capsule. It was only when the patient began walking around that blood escaped from the small wound in the spleen, to produce all the symptoms of internal hæmorrhage.

Editorial

HEALTH INSURANCE IN BRITISH COLUMBIA

WE are giving special prominence in this issue (see page 297) to an account of the situation in British Columbia regarding its Health Insurance problem. No member of our Association should fail to make himself acquainted with what is going on in that Province. Time and again it has been said in these columns that our profession is being steadily and inexorably drawn into vitally altered conditions of work. Now we are witnessing a crisis in this movement. The Provincial Legislature in British Columbia has set up a Health Insurance Act which is to come into force in a few weeks, but the plan of its operation *has never received* the approval of those who will be called on to carry it out, namely, the medical men of the Province. At the time of going to press there is still no final decision, but as will be gathered from our "Association Notes" there is a ballot in progress throughout the Province which will show the attitude of the profession towards the Act. A preliminary, but unofficial, vote at a mass meeting in Vancouver has shown an overwhelming majority in opposition.

Need we dwell on the gravity of the situation? We can at least reiterate our sympathy for those of our profession who are carrying on this painful struggle for proper recognition. We must also express our admiration for the patience and dignity with which the representations of the medical men have been made in the face of political forces determined, to put the mildest construction on it, on exacting services for intolerably inadequate returns.

H.E.M.

RHEUMATIC FEVER AS AN INFECTION

AFTER a perusal of the recent literature on rheumatic fever one turns away with a feeling of disappointment and dissatisfaction. It is now thirty-six years since Poynton and Paine announced the discovery in the blood of patients with acute rheumatic fever of a streptococcus which they considered to be the cause of the disease. Since then studies of the disease have been extensive and intensive. They have included observations on cultures from the pharynx, blood, and joint tissue; on agglutinins, precipitins, opsonins and streptolysins in rheumatic and non-rheumatic patients; on skin tests; on the histology of the various lesions; and on the experimental production of arthritis in animals. In spite of all this there is no unanimity of opinion as to the nature of rheumatic fever. Every possibility in regard to etiology and pathogenesis has been canvassed and every theory has been controverted. We seem, indeed, to be very little nearer the complete solution of the problem. Possibly the reason for this is a lack of proper method.

There are three main theories as to the causation of rheumatic fever: first, the infectious theory, with its variants; second, the endocrine theory; and, third, the metabolic theory. Doubtless, alterations in the body metabolism occur in rheumatic fever, as in many other conditions, but they are more likely to be an effect than a cause. The endocrine theory is too nebulous to

detain us at the present time. We may safely say that the majority of observers are adherents of the first view in some form or other.

There can be little doubt, despite some evidence to the contrary, that rheumatic fever is an infection, if not, indeed, narrowly specific, at least broadly so. Its clinical course, with pharyngitis or tonsillitis, fever, leucocytosis, arthritis, the involvement of various serous membranes, endocardium, pleura, peritoneum, and meninges, the unequivocal signs of inflammation in the observable lesions; and the widespread distribution of an apparently specific lesion, the "Aschoff body", are strong positive evidence. This conception implies the dissemination through the body of a micro-organism which gains entrance to the blood from some focal point, usually the throat. As regards possible infective agencies these may be (*a*) one specific strain or group, (*b*) organisms that are group- but not strain-specific, or, (*c*) any one of a number of different organisms. To prove the truth of this view we need to have positive blood cultures, preferably of an individual type or strain of organism in a significantly high percentage of cases. So far, such proof has not been consistently afforded. A few of the pertinent studies may be cited. Gray, Fendrick and Gowen[1] isolated streptococci, usually viridans or of alpha prime type, in 71 per cent of 28 cases of rheumatic fever. It is worthy of note that in two cases hæmolytic streptococci were isolated early in the attack and later on *S. viridans*. One hundred and thirty-two cultures in 105 pathological controls were negative except in 15 instances where positive blood cultures were to be expected. Wilson and Edmond[2] obtained positive cultures from the blood in 46 per cent of 67 cases of acute rheumatic polyarthritis in children, and in about the same percentage in the case of children who were ill of troubles other than rheumatic fever, and in a group of healthy children. About half of the organisms obtained were streptococci, either viridans or indifferent forms; the others were pleomorphic bacilli. It may be remarked here in passing that the writer, in examining smears from throats, has frequently noted that some members of a chain of streptococci assumed a bacillary form. Therefore, it should not be hastily concluded that bacillary forms found in the blood cannot be streptococci. Wilson and Edmond concluded that bacteria may gain access to the blood stream of both healthy and sick children and are filtered out in various organs, where they are destroyed. The presence of certain bacteria in the blood of rheumatic children would not seem to be of primary etiological importance in their opinion, unless, of course, we postulate that the tissues of the rheumatic child are specially sensitive to the organisms present in their blood. Allergy as well as bacteriæmia may be invoked here. Callow[3] obtained about the same type and number of blood cultures in two groups; in 53 per cent of 174 patients with rheumatic fever; in 66 per cent of 58 persons with acute affections of the upper respiratory passages; in 34 per cent of patients with miscellaneous diseases; and in 8 per cent of 39 normal persons. The organisms isolated from the rheumatic group were *S. viridans*, non-hæmolytic streptococci, and pleomorphic bacilli. It is significant that Callow was able, under controlled conditions, to transform selected strains of the bacilli into diplostreptococci of either viridans or hæmolytic type, similar in morphological, cultural and biochemical characters to the diplostreptococci isolated from rheumatic and non-rheumatic patients. He questioned whether there was a specific etiological relationship between the organisms isolated and rheumatic fever. Similarly, Rosenow, by altering environmental conditions, was able to transform viridans forms into a hæmolytic or indifferent type. Identification of types is therefore difficult and confusion arises. Cooley[4] found no significant organisms in the blood of 25 children in initial attacks of rheumatic fever.

In conclusion, it may be added that aggluti-

1. GRAY, J. W., FENDRICK, E. and GOWEN, C. H.: Rheumatic fever and rheumatoid arthritis from laboratory point of view, *Texas State J. of Med.*, 1932, **28**: 317.
2. WILSON, M. G. and EDMOND, H.: Blood cultures in children with rheumatic fever, *Am. J. Dis. of Child.*, 1933, **45**: 1237.
3. CALLOW, B. R.: Bacteriologic investigation of the blood in rheumatic fever; presenting evidence of dissociation of micro-organisms recovered from blood cultures, *J. Infect. Dis.*, 1933, **52**: 279.
4. COOLEY, L. E.: Blood culture in rheumatic fever, *J. Infect. Dis.*, 1932, **1**: 330.

nation and precipitin tests have proved inconclusive, at least in so far as incriminating a particular form of streptococcus is concerned, nor have attempts to reproduce rheumatic fever in animals always proved successful.

It is clear that the final proof that any particular micro-organism is the specific cause of rheumatic fever is still wanting. Possibly, what is needed is that the various investigators should settle on some standard method for culture of streptococci and all adhere to it. The possible variations in the strains obtained in regard to morphology, colour production, hæmolysis, morphology and virulence should then be thoroughly explored. It would seem to us to be essential that the bacterial content of the blood in rheumatic fever should be determined daily, and also the character of the bacterial output, if any, in the urine. We may take, by way of illustration, the case of typhoid fever. The causative germ was known for long, but it was only when relatively large quantities of blood (10 c.c.) were used for culture, and the cultures were made every day, that the sequence of events was made out. The *Eb. typhosus* is to be found in the blood during the first three to seven days; it gradually disappears from there, to be found in the spleen and bile; then it may be found in the stools, when the sloughs are separating; and, finally, it may be excreted in urine for weeks and months, and in the bile for months and years. Of course, in the case of rheumatic fever the problem is probably not so simple. The *Eb. typhosus* is confined to the human species, is only occasionally found in nature (in water, milk, and other foods), and has fairly constant properties; the streptococcus is found in the lower animals as well as man, is ubiquitous, is, in some forms at least, parasitic and not pathogenic, and is inconstant in its features. But this only means that the methods of research should be better standardized, be more comprehensive, and more searching.

A. G. N.

THE OPERATIVE TREATMENT OF CARDIAC ISCHÆMIA

THE most important results of ischæmia of the cardiac muscle are myocardial fibrosis and its congener, coronary thrombosis. The latter condition is so spectacular in its manifestations that it at once rivets attention and its diagnostic features are known to all. Up to the last three or four years the treatment has been entirely medical, and, in the main, symptomatic and palliative. We rely on morphine and the barbiturates to relieve pain and promote rest, on measures to combat shock, on appropriate diet, on absolute quiet in bed, and, for the rest we trust in Providence. Comparatively little thought is being given to other measures for restoring the heart to adequate function. Chronic myocardial fibrosis and, still more, coronary thrombosis lessen the supply of blood to the heart muscle and so reduce its effectiveness; and the need of the heart for blood is great, even under normal conditions. It has been stated that during exercise the coronary arteries convey 1.4 litres of blood per minute, the volume of which is equivalent to three times the size of the heart; and a fall of 50 per cent in oxygen saturation of the blood would require the coronary flow to be increased fourfold in order to compensate for it. Such facts serve to bring home to our minds the importance of maintaining an adequate circulation of blood in the cardiac muscle and also the great demands that are to be required of any remedial measure.

Basing themselves on experimental evidence, Fowler, Hurevitz and Smith,[1] and Smith, Rathe and Paul[2] have reported encouraging results in coronary occlusion from the use of theophylline ethylendiamin and other members of the theobromine group, such as theocin and diuretin, which drugs apparently act by favouring the production of a collateral circulation. While such reports are encouraging, we must await further experience before assessing the value of these medicinal agents. Surgical measures are occupying the limelight at the moment,

1. FOWLER, W. M., HUREVITZ, H. M. and SMITH, F. M.: The effect of theophylline ethylendiamin on experimentally produced cardiac infarction in the dog, *Arch. Int. Med.*, 1935, **56**: 1242.

2. SMITH, F. M., RATHE, H. W. and PAUL, W. D.: Theophylline in the treatment of disease of the coronary arteries, *Arch. Int. Med.*, 1935, **56**: 1250.

and it is to these that we wish to direct attention now.

A case is known (that of Thorel[3]) in which, at autopsy, complete obliteration of both coronaries was found. There had been no symptoms referable to this during life, and the situation seemed to have been saved by the presence of vascularized pericardial adhesions. Thus was suggested to C. S. Beck, of Cleveland, Ohio, the propriety of investigating experimentally the possibility of producing a similar condition of compensation by surgical measures. The relative papers are noted below.[4,5,6,7] Beck has operated on 11 patients with coronary sclerosis and angina pectoris, all of them being bad surgical risks. Six are living and five are dead. Five reported improvement and one result was doubtful. He used grafts on the pericardium of experimental animals of skeletal muscle, pectoralis major, triangularis sterni, intercostal muscle, and omentum, and demonstrated the presence of anastomoses in the grafts after three weeks. He has not grafted omentum on the pericardium in the human subject, as he thinks it adds certain undesirable complicating features.

Laurence O'Shaugnessy, of London, began his investigation of the subject of pericardial grafting at the experimental station at Downe, in 1933. He has described[8] the first results of a series of grafts in cats and, more particularly, in the greyhound, showing that the existence of such grafts is compatible with the highest degree of physical exertion. The greyhound was eventually killed, to prove that the anastomosis got better with time and not worse.

The rationale of cardio-omentopexy is based upon the following considerations. The omentum is the only structure in the body that has the specific power of vascularization. The Drummond-Morison operation, or its modification by Talma, which consists, essentially, in suturing the omentum to the abdominal wall for the relief of ascites in cirrhosis of the liver, is well known and its value has long been recognized. Long ago, also, Sir David Wilkie showed that in the cat the results that followed the ligation of all the vessels going to the spleen could be prevented by attaching the omentum.

Mr. O'Shaugnessy, in a special lecture recently delivered before the Royal College of Surgeons of England,[9] described his method of performing cardio-omentopexy in cases of coronary disease and gave his results. He was of the opinion that the graft operated not only to bring a new blood supply to the ischæmic area of the coronary tree but also reinforced the natural collaterals in the mediastinum, giving them access to the devascularized part. Two considerations guided him in making a decision to operate. The first was that the evidence in favour of a condition of cardiac ischæmia must be convincing, and the second, that the immediate risks of operation must seem to be less than those the patient would run if his trouble were allowed to pursue its natural course. Mr. O'Shaugnessy thought it of great importance that the operation should be such as to disturb as little as possible normal function and that surgical shock should be avoided. In six cases on which he had operated four patients seemed to have benefited. One died of hæmorrhage from a duodenal ulcer and one succumbed later to uræmia. The latter had had some relief for a period from his angina. One patient left hospital and, apparently, could not be followed up. Mr. O'Shaugnessy, in referring to Dr. Beck's operation, in which a pedicle graft from the pectoralis major was sutured to the heart, stated that he felt that in this case the factors concerning shock could not be so readily controlled as in the case of cardio-omentopexy.

Beck[7] has doubts as to whether grafts of skeletal muscle are quite safe, as large muscle grafts may be a factor in causing arterial thrombosis through the liberation of tissue juices into the blood stream. He is hoping to get away from the major operation in

3. THOREL, C. H.: Pathologie der Kreislaufsorgane, *Ergebn. allg. path. u. path. Anat.*, 1903, **9**: 559.
4. BECK, C. S. and TICHY, V. L.: The production of a collateral circulation of the heart, *Am. Heart J.*, 1935, **10**: 849.
5. *Ibid.*: The production of a collateral circulation to the heart, II. *Am. Heart J.*, 1935, **10**: 874.
6. BECK, C. S.: The development of a new blood supply to the heart by operation, *Ann. of Surg.*, 1935, **102**: 801.
7. *Ibid.*: Further data on the establishment of a new blood supply to the heart by operation, *J. Thoracic Surg.*, 1936, **5**: 604.
8. O'SHAUGHNESSY, L.: An experimental method of providing a collateral circulation to the heart, *Brit. J. of Surg.*, 1936, **23**: 665.
9. O'SHAUGHNESSY, L.: The treatment of cardiac ischæmia, *Brit. M. J.*, 1937, **1**: 184.

cases of cardiac ischæmia, and is trying out the introduction of powdered bone into the pericardial cavity. This procedure is known, in the case of experimental animals, to be followed by pericardial synechia. If fluid collects in the pericardial sac as a result it is easy to tap. It is hoped that some such simple measure may be discovered which will be as effective as muscle or omental grafting, less dangerous, and more widely applicable.

The clinical course and prognosis of coronary thrombosis are so variable and uncertain that it is evident that the applicability of operative measures for its relief is quite limited. Obviously, operation would be contraindicated during the acute stage of an attack, or if serious decompensation of the heart has set in. One might suggest that if an x-ray film showed the presence of an aneurysm of the heart wall a graft would be of value in providing a material buttress to the weakened structure. The general criteria by which Mr. O'Shaugnessy guides himself, as given above, would seem to afford a safe rule, at least until further experience with the operation has been recorded. A. G. N.

Editorial Comments

The Late Sir John Bland-Sutton, Bart.

We regret to have to announce the death on December 20, 1936, of the eminent surgeon Sir John Bland-Sutton, Bart.

Sir John entered Middlesex Hospital, London, as a medical student and obtained his M.R.C.S. in 1880 and F.R.C.S. in 1884. He was appointed to the staff of the hospital and became successively Lecturer in Anatomy, Assistant Surgeon, Surgeon, and, finally, in 1920, Consulting Surgeon. He was one of the first in that hospital to undertake abdominal operations, in the early days of Listerian asepsis. In 1896 he was appointed Surgeon to the Chelsea Hospital for Women. In 1910 he was elected to the Council of the Royal College of Surgeons and was its president for 1923-25. He was president of the Medical Society of London in 1914. He was the first president of the Association of Surgeons of Great Britain and Ireland, which was founded in 1919, and in 1920 became president of the Royal Society of Medicine.

Sir John was knighted in 1912 and received a baronetcy in 1925. He was a Knight of Grace of the Order of St. John of Jerusalem, and held the LL.D. degree from the Universities of Aberdeen, Birmingham, Glasgow, Leeds, and St. Andrew's; D.Sc. of Toronto; M.Ch. of Trinity College, Dublin; and M.D. of Bordeaux.

Sir John contributed much to medical literature, and was the author of several well known books. Chief among them are "Evolution and Disease"; "Tumours, Innocent and Malignant"; "Gallstones and Diseases of the Bile Ducts"; "Selected Essays and Lectures"; "Diseases of Women"; "Man and Beast in Eastern Ethiopia"; "The Story of a Surgeon". In his work on tumours he endeavoured to set forth a classification on embryological principles and to illustrate his points by comparative pathology. In his interest in the lower animals and his love for scientific method Bland-Sutton suggests comparison with John Hunter. As a surgeon he was progressive and resourceful; as a speaker he was distinguished above the average. A.G.N.

The Late Professor D. F. Fraser-Harris

The former associates and many friends in Canada of Professor Fraser-Harris will regret to learn of his demise, which took place in London on January 3rd.

Fraser-Harris was born in Edinburgh in February, 1867, the son of the late David Harris, F.R.S.S., F.R.S.E., of Lyncombe Rise, Bath, England, and Elizabeth Sutherland Fraser, of Fort William, Scotland. He was educated at the Edinburgh Collegiate School, Edinburgh and Glasgow Universities, and University College, London. He pursued postgraduate studies also at Bern, Jena, Zürich, and Heidelberg. He held the following degrees: M.B., C.M. (1893); M.D. (Glas., 1895); B.Sc. (Lond.); D.Sc. (Birm.); and was also a Fellow of the Royal Societies of Edinburgh and Canada. Before coming to Canada he was Muirhead Demonstrator of Physiology at Glasgow (1893-98); Acting Professor of Physiology, University of St. Andrew's (1898-1908); Lecturer in Physiology, University of Birmingham. He was appointed Professor of Physiology in Dalhousie University, Halifax, N.S., which post he held from 1911 until his retirement in 1924.

During his Canadian period Professor Fraser-Harris made his mark as an able and far-seeing investigator in his subject, and was highly esteemed as a finished lecturer on scientific and popular topics. He was well versed in the history of medicine and had made a special study of William Harvey. Besides these activities he entered usefully into the life of his com-

munity. He was, among other things, Adjutant of the Dalhousie Contingent, C.O.T.C.

Perhaps his chief contribution to physiology was his enunciation of the doctrine of "physiological inertia and momentum". This conception had been put forward previously by Weigert, but Fraser-Harris was the first to state it in clear terms. This he did in his "The Functional Inertia of Living Matter" (London, Churchill, 1908). Other works of his are the following: "The Official History, Medical Aspects of the Halifax Disaster" (1917, unpublished; now in the Archives Dept., Halifax, N.S.); "Life and Science" (1923); "Nerves, the Master-System of the Body" (1927); "Coloured Thinking" (1928); "The Sixth Sense" (1928); "Morpheus, or the Future of Sleep" (1928); "The ABC of Nerves" (1928); "The Rhythm of Life" (1928). He was also editor of The Modern Health Series. In all his writings Fraser-Harris evinced a comprehensive grasp of his subject and presented his points convincingly and attractively. He was a master of English.

Professor Fraser-Harris is survived by his widow, formerly Eleanor Leslie, daughter of Lieut.-Col. F. M. Hunter (Bombay Army), and by one son, now an officer in the Royal Navy.

A.G.N.

The State of Scientific Literature

The difficulties connected with scientific publications are increasing, although it is doubtful if many realize their significance, or even that they exist. Those most likely to do so are the comparatively small groups of research workers, of librarians, and of editors, and it is the librarians who, not unnaturally, have the most to say about it. Their task it is to house the mounting mass of printed material, and to make it most accessible to those who would explore in it. A particularly interesting commentary on the situation was made at the last meeting of the Medical Library Association, by Mrs. E. R. Cunningham, who brought up three main factors for consideration: (1) the increasing quantity of material being brought forward for publication; (2) the difficulties connected with the abstracting of what is published; (3) the enormous number of journals being produced.

Mrs. Cunningham's remarks apply to scientific literature as a whole, but the same problems exist in regard to medical literature, and present no greater tractability. We too in medicine have too much material sent in for publication; we too wish for a more comprehensive method of abstracting what is published; and we too suffer from excessive journalism.

It was suggested that more use be made of the recently perfected method of microphotography, by which the material to be recorded is photographed on to small areas of film, which can be enlarged by projectors whenever needed. This method has already been put into practice to a limited degree, and has great possibilities in allowing the transport of large quantities of printed material to otherwise inaccessible places. It also provides for storage in negligible space of what otherwise would be unwieldy. But this plan does not by any means solve the problem. For one thing, it calls for a degree of centralization and cooperation which is a very long way from attainment in the scientific world. Also, it would tend to supplant many scientific journals, if carried to a logical conclusion. The most severe criticism of it probably came from the librarians themselves. Their conclusion was that it would be much better to have fewer and better papers and have them printed, rather than yield to the pressure of undiscriminating output. In any case, we are not yet ready to discard books and articles in the printed form.

Another point brought up at this meeting was the apparent demand for a clinical abstract journal. It was shown that at the present time there is a large amount of duplication of abstracting in the various journals. The same paper may be abstracted by three or four journals, and since there are 146 journals at present which carry abstracts, these obviously imply a large expenditure of time and money and space, much of which might be eliminated. In addition, readers of journals do not even get consistently thorough reviews of what is worth while. It is suggested that some enterprising publishing house should try and bring out some publication which would include all the required abstracts under one cover, but apparently the difficulties so far have been too great.

H.E.M.

The Late Dr. D. A. Stewart

It has been known to all his many friends that Dr. D. A. Stewart, of Ninette, Manitoba, for months past had not only been suffering the extremities of a grave and painful illness, but had also been overtaken by an overwhelming tragedy in the loss of his wife. Our concern and deep sympathy followed him continually. There is no one who has served Canadian medicine more faithfully and ardently, both through his own immediate occupation in fighting tuberculosis, and also through his long and intimate connection with Association activities. At the time of his illness Dr. Stewart was engaged in coordinating the work of the Committee on Ethics, or, rather, was initiating and carrying out most of its labours himself. Up to a short time ago he had managed, as only one of his spirit could, to keep up his interest in affairs, but the draining away of his strength was all too evident and he passed away within the last few days.

We have lost a great man, who never failed in his devotion to public duty.

H.E.M.

Corrigendum

Dr. J. S. L. Browne writes to say that there is an error in the abstract in the *Journal* (1937, **36**: 83) of the paper entitled "The Measurement of a Pregnandiol Complex in Human Urine", by Eleanor M. Venning, J. S. Henry and J. S. L. Browne. The sentence "During pregnancy the output in the early months has not yet been definitely determined, but there is a definite increase in the rate of excretion of this substance, which begins usually late in the *second* or early in the *third* month", should conclude "which begins usually late in the *third* or early in the *fourth* month".

Medical Economics

The Situation in British Columbia Regarding The Health Insurance Act

(We give below an exact transcript of what appears in the February issue of the *Bulletin of the Vancouver Medical Association* regarding the plan suggested for the carrying out of medical practice under the Health Insurance Act. The material consists of (*a*) An Interim Report of the Health Insurance Committee of the College of Physicians and Surgeons of British Columbia; (*b*) an analysis of the plan suggested by the Health Insurance Committee; (*c*) the Editorial comment of the *Bulletin* on the situation.

We feel that this deserves the closest attention of all our readers.—ED.)

HEALTH INSURANCE COMMITTEE, COLLEGE OF PHYSICIANS AND SURGEONS OF BRITISH COLUMBIA

INTERIM REPORT

On January 20th a very full meeting of the Health Insurance Committee of the College of Physicians and Surgeons of British Columbia was held at Vancouver, and lasted four and a half hours.

The members of the Committee are as follows: Dr. T. McPherson, Chairman; Drs. Wallace Wilson, W. E. Ainley, L. H. Appleby, J. A. Gillespie, J. J. Gillis, B. D. Gillies, A. W. Hunter, G. C. Kinning, R. L. Miller, H. H. Milburn, J. H. MacDermot, S. C. MacEwen, W. H. Sutherland, C. H. Vrooman, M. W. Thomas.

Supplementing this Committee were representatives from all parts of the Province, as follows: Drs. G. F. Strong, W. T. Ewing, A. J. MacLachlan, R. J. Wride, E. W. Wylde, R. B. White, J. H. Hamilton, O. O. Lyons, F. M. Auld, F. P. McNamee, F. W. Green, J. Bain Thom, C. H. Harkinson, G. A. B. Hall, E. J. Lyon, R. McCaffrey, H. C. Graham.

It will thus be seen that the group was a very representative cross-section of the entire profession of British Columbia.

The Tentative Plan of Medical Practice under the Health Insurance Act was considered and discussed clause by clause by this group. A copy of this plan will be received by every member of the profession, within the next few days, from the Health Insurance Commission. In all centres, meetings will be held within the next week or two, where representatives from the group referred to above will be prepared to discuss all questions that may be raised.

The group considered also: (1) the regulations that would govern the general practitioner; (2) the regulations that would govern the insured; (3) a list of exemptions prepared by the Commission, to be in force for the first year or so. Letters between the Commission and the Committee were considered, and after a full discussion, it was unanimously decided that the plan as outlined could not be accepted as satisfactory.

On January 20th this enlarged group met the Health Insurance Commission, and had a long discussion with them on the subject of the Tentative Plan. The Chairman of the Commission answered freely and fully all questions that were asked. The group held a second meeting by itself to consider the suggestions made, and again unanimously decided that we could not regard the plan as satisfactory. Later in the evening, we again met the Commission. Dr. T. McPherson, the President of the Council of the College of Physicians and Surgeons of British Columbia, and Chairman of the Health Insurance Committee, then addressed the Commission.

He pointed out to them: First, that, as a committee, we could not accept or reject the proposals of the Health Insurance Commission, but must refer them to the profession for their vote; second, that, as a committee, we were of the opinion that the Act as at present proposed was unsatisfactory, for the following reasons:

I. As regards the insured:

That the Act gives insufficient service, in that it does not include the indigent, or make any provision for them or the people on relief; it does not include: old age or mothers' pensioners; domestic servants; casual and part-time labourers; those in receipt of less than $10 per week.

Hospitalization as offered under the Act is impossible of attainment under present conditions, or for a long time to come, as there is no possibility, in any of the larger, and most of the smaller, centres of the province, of providing enough accommodation for any increase.

Mileage is not paid for, and will have to be charged to the patient by the doctor.

II. As regards the profession:

We regard the remuneration proposed as insufficient for the general practitioner, secondly for the specialist and consultant. All our studies, and all studies made all over the continent, show that the fund proposed is not adequate by a considerable amount. The Commission will not allow any choice of payment, but insists on the capitation fee for doctors.

We estimate that the amount of work demanded of the doctors will be greatly increased, and that this, too, is an important factor.

Dr. McPherson, further speaking for the Committee, assured the Commission that we were prepared to recommend to the profession that they be willing to work under a partial service for the first year, such as, for example, a system that would provide hospitalization, drugs, and complete diagnostic methods; or any other scheme that might be devised jointly by ourselves and the Commission which would give a service commensurate with the amount of money available.

The Commission, on the other hand, urged that we try the scheme for a year, and that at the end of that time, if funds were shown to be inadequate, they would be willing to go to the Government and recommend one of the following plans, or any combination of two or more: (1) increase of assessment; (2) decrease of benefits; (3) a subvention by the Government to the fund.

Your Committee felt that this was entirely too vague and uncertain, and meant that the medical profession had to assume all the risks, and make all the sacrifices, while we also feel that these suggestions are of very doubtful value, unless definite promises can be obtained before the Act is put into operation.

The Commission would only undertake to make an attempt to secure such promises on the condition that the profession agree to make trial of the Act for a year.

At parting, the matter was left open for further negotiations.

A BRIEF ANALYSIS OF THE TENTATIVE PLAN SUGGESTED BY THE HEALTH INSURANCE COMMITTEE

1.—THE DISTRIBUTION OF MEDICAL SERVICES

On the whole, there is room for only minor criticisms here; questions of detail arise, especially with regard to specialists: *e.g.*, roentgenologists working in hospitals may receive cases either from the hospital, or referred from outside; the method of payment is not clear, whether from the hospital, the specialist pool, or other funds.

Exemptions. Here there is a question of great importance. First, it must be clearly pointed out that this list of exemptions was prepared on the insistence of the Commission, and in face of the protest of the Committee, who felt that, in a proper Health Insurance Scheme, there should be no exemptions. The Committee felt, too, that if exemptions were laid down, the actual wage-earner should be free of them, and as regards the dependents, only those conditions which could have been diagnosed and treated to a cure before Health Insurance came in should be included.

The Commission, on the other hand, stated frankly that they felt exemptions were necessary to prevent excessive cost, especially the cost of hospitalization.

The actual list of exemptions contains some conditions that, we feel, are bound to lead to conflict, *e.g.*, post-partum conditions. Here the possibility of cancer in later life, and of other conditions arising, is going to lead to much confusion and discussion.

The diabetic, too, cannot obtain insulin, or the pernicious anæmia patient liver extract, as part of the free benefits.

From our point of view this list of exemptions promises to lead to much trouble, as has been found to be the case in many places where contracts are in force, and attempted exemptions have caused trouble—nor has the doctor been able to collect payment for them.

2.—THE METHOD OF PAYMENT FOR MEDICAL SERVICES

It is in this part of the plan that the chief differences arise between the Commission and ourselves.

The charge of $1.00 or $1.50 for first house calls in any one illness is an innovation, we feel, that is not satisfactory. In the first place, the insured will resent it greatly, and, we cannot but feel, with considerable justice. This is in direct contradiction to the principle of a complete service.

In the second place, there can be no doubt that it will be very difficult to collect, though we must acknowledge that the Commission has shown its desire to cooperate in the methods it

has suggested to make this fee a reality. But these methods, while they may work, can be evaded, and there is no definite, certain method of collection devised; and in places where some such scheme has been tried under contract practice, it has always failed, after proving to be a fertile source of trouble.

The method of payment, by capitation fee for the general practitioner, by payment from a fixed pool of taxed bills for the specialist.

The capitation fee method of payment is, we believe, a bad method, based on a wrong principle. The medical profession of Canada has always opposed it. We cannot here go into detail—but we again record our settled opinion that, as has always been our rule, definite service should be paid for on the basis of a definite schedule of fees, as is done by the Workmen's Compensation Board and in private practice. We still adhere to this principle.

Next, as regards payment from a fixed pool. This is a most pernicious principle, as we believe. The specialist does the work, renders his bill according to the scale of fees adopted by the British Columbia Medical Association, and then has no means of knowing what percentage of his bill he will receive—since this depends on the ratio of the total amount of bills sent in to the fixed pool. We have no hesitation whatever in saying that this is entirely wrong. No man in any other walk of life would undertake to do work without knowing how much he could be sure of receiving—no business man would sign any such contract as this. It is none of our business to tell the Commission where they are to get the money—but we have a right, before we undertake work, to know how much we shall receive.

As regards the total available for general practitioners—$4 for those who do their own surgery, $3.60 or more for those who do none or part, and so on.

We regard this as quite inadequate. In the first place, this represents the gross income: *i.e.*, for 1,000 patients the medical man may receive $4,000 gross, plus allowance for maternity patients at $24 each, plus a quite unknown amount for first house calls, plus a possible amount for exempted diseases, plus any consultations or special work he may do. Workmen's Compensation fees continue as at present.

Apart from the Workmen's Compensation Board, the general practitioner cannot possibly receive more than $4.35, plus the problematical amounts for first house calls and exemptions and consultations, plus a share of the 50 cents extra allowed for contingencies.

Very careful studies have been made all over the world, and one of the most thorough computations of all has been made in Vancouver. From every study, including those of the Committee on the Costs of Medical Care, the studies in Michigan, in Ontario, here and elsewhere comes a figure which is roughly the same everywhere, namely $7.30 to $7.50 for a complete medical service, including Workmen's Compensation Act, specialists, etc. Workmen's Compensation fees average, we believe, about 50 cents per capita, certainly not much more.

So there is evidently a wide discrepancy, and nowhere in the world is $5.50 accepted as a fair figure—nor do we believe that it begins to give adequate remuneration.

All these points have been made clear, again and again, to the Commission, to no avail.

We know, too, that under any scheme of Health Insurance work has always greatly increased—in fact, it is evident from the Preamble to the original Brief of Dr. G. M. Weir that a very great increase will be expected from us.

As regards specialists, the fund provided is quite inadequate, in our opinion.

Mileage.—We must further point out that the Commission has definitely declined to make any allowance for mileage, which must be collected by arrangement between the doctor and the patient. This is grossly unfair to the latter, and in fact will, we believe, make service under this scheme impossible in large areas of the Province.

From the standpoint of the Insured.

The Committee feels, and so informed the Commission, that it cannot consider this Act adequate from the public standpoint.

In the first place, it omits all the very people in the community who most need medical care—the indigent, those on relief, domestic servants, old-age and mothers' pensioners, casual and part-time labourers, and those earning less than ten dollars a week.

No provision is made for these at all, and the medical profession must still continue to carry this whole load of unpaid work, both in and out of the hospital, without any compensation whatever.

After we had made many requests for action along this line — which, in our belief, was promised again and again, with no result—we finally obtained a statement from Premier Pattullo that the question of the indigent would be taken up and settled *after the Health Act was in force.* We frankly cannot accept this as of any value at all.

Secondly, the service given is not complete. The list of exemptions is the first break—and denies essential treatment to certain groups, *e.g.*, the diabetic, the patient with pernicious anæmia, and to certain women who need surgical attention.

The charges for first calls are an additional tax to the man with a family, and may constitute quite a burden.

Hospitalization.—This is, in our opinion, a very serious matter. Every hospital in British Columbia, almost without exception, is now filled to capacity. In such centres as Vancouver there is already a grave shortage, of some 500 to 600

beds. It is quite certain that when Health Insurance comes in there will be a great increase in hospitalization—far more than the Commission will allow will be the case. Further, the experience of Australia and other places shows a great increase in hospital days for each patient under free plans.

Free hospitalization is promised, and is one of the chief elements in the plan.

We do not believe that there is any possibility of giving it under present conditions.

Mileage.—This, as shown above, will also be a tax on the insured that is unfair and unjust.

If insufficient remuneration is paid to the medical profession, good work cannot be expected, and this is bound to react on those receiving benefits. We believe that under a cheap, underpaid scheme, the quality of medical service given is bound to be low, and to deteriorate—and the chief sufferers will be the insured.

The medical profession is not opposed, as we assured the Commission, to Health Insurance. Rather, it is in favour of it, almost unanimously; but there are certain principles which we feel must be observed, amongst them a fair and adequate remuneration for the medical man, on scales which conform with the findings of all the major surveys. We believe, too, that payment should be by a definite fee for definite work, not by capitation fee. That service given should be complete and inclusive, without exemptions, if a complete scheme is to be inaugurated—and if this is to be the case, sufficient money should be provided. We feel that if there is not enough money to provide a complete scheme, under terms fair to all concerned, then a partial scheme should be installed at first, under which we could feel our way safely and without injustice to any, towards a more ample scheme later. We oppose strongly the idea that the medical profession should take all the risk, but we reiterate our complete willingness to explore all avenues towards the establishment of a fair and practicable scheme, and will prepare and suggest alternative plans to that end, or consider honestly any that may be brought forward. But, as this plan stands at present, we cannot feel that it is satisfactory, or recommend it to the general profession.

EDITORIAL

We are undoubtedly at a very vital crossroads in the history of medicine, not only in British Columbia but throughout Canada. It behooves us all to think gravely, and carefully, before we act. Not temporary expediency, and not apparent self-interest, must guide us, for what may seem of benefit at the moment may later turn out to be the first step to suicide.

Nor must we let ourselves be influenced by defeatists and those who would urge us to a *sauve qui peut* policy. Thank God, there are few of these in our ranks, but there are some, and we must not give them undue ear.

Nor need we yield to fear. We have been told in no uncertain terms that the end of bargaining has been reached—that we can expect no further concessions. Further, we are advised to accept the terms that are offered, as otherwise there will be a fight, and, we are told, a bitter one. So we come to the end of nearly three years of what may be called bargaining, if by bargaining we are to understand a state of affairs where one side offers less and less, and the other is expected to yield more and more. Poor as the first Act was, it was immeasurably ahead of this one, and baits held out have been withdrawn one by one till there is little in the poor fragment left to attract the hungriest fish.

One or two outstanding considerations emerge from this long struggle: The first is that expediency and not statesmanship, political profit and not service to the state, have gradually cheapened and weakened what was in its inception a noble scheme, fraught with great promise of good, and demanding the sympathetic cooperation of our profession. But the present scheme is a miserable patchwork. Those who really need medical care most are completely omitted from its provisions; those who are dubbed "beneficiaries under the Act" will not get the full service they have been led to expect.

We are asked to do a greatly increased amount of work for a sum which every survey that has ever been made shows to be grossly inadequate. The Committee on the Cost of Medical Care, the Michigan inquiry, the work in Ontario, in every other locality, and our own records, compiled through the excellent work of Dr. W. E. Ainley, all come to exactly the same conclusion, within a few cents, as to what constitutes a fair remuneration for medical men. The British Columbia scheme offers a sum so far below that it means totally inadequate remuneration.

As for the consultant and specialist—these are asked to send in bills according to the schedule of fees of the British Columbia Medical Association; but what share of these they receive will depend, not on this valuation, but upon the proportion that the total bears to the amount in the fixed pool, which cannot be exceeded. No business man would consider for one moment signing any contract on such terms; no member of the Commission would ever consent to work under such conditions as that his salary would depend, not on agreement beforehand, but on the amount of money available after all expenses had been met. Nor should we agree to work on any such conditions.

We are asked to trust the Commission, and their assurance that if means are found inadequate after a year's trial, they will en-

deavour to have this remedied. In other words, we are to try it for a year and then see. This is altogether too much like the spider's invitation to the fly. No, we must realize that when we take this step there will be no turning back. Increase of assessment, reduction of benefits, government grants, in our interest, are just so many airy dreams. They would be fought so bitterly that we should stand no chance of any success on those lines.

The medical profession has laid down, again and again, the cardinal principles which it believes should govern Health Insurance. It believes in Health Insurance, it will agree to work under it, and at a sacrifice to itself—but it must not be asked to make all the sacrifice, take all the risk, and do all the work. We have a perfect right to bargain—to dispose of our services at a fair figure. We believe that we are in the right in this matter, and that since our quarrel is just, our armour is of triple might. We should know, before we enter this scheme, which depends entirely on the medical man's work, what we shall be asked to do, and how much we shall be paid for our work. The Act does not answer either of these questions.

Anyone who has sat in at the deliberations, month after month, of the various representatives of the profession must feel, as we feel, intensely proud of the loyalty and true worth of his fellows. Throughout the discussions, the note of what constitutes a good Act from the point of view of the public has been struck; it is quite safe to say that self-interest has been quite secondary; that the Committee could not sooner let the profession know all that was going on was due to unavoidable conditions of ignorance on its own part—it is only within a very short time that the Committee itself knew the final suggestions of the Commission. Meantime, we feel that the greatest praise is due to the members of the profession who have loyally backed up their Committee, and, in the most trying conditions, have given them a confidence which alone has made it possible for them to carry on.

We feel that we understand the temper of the medical profession today; that it will still hold itself ready to work with the Commission and the Government in any fair and worthy scheme that will give a real and good medical service to those who really need it, at a cost which can be afforded by those who have to pay, and on terms of payment to those who work under it which are fair and equitable. We believe that the vast majority will oppose any scheme which does not do this, and not alone from selfish motives—though we believe, too, that we are entitled, as are all people who must work for pay, to the usual considerations that apply to every other job under the sun—definite pay for definite work.

The Responsibility of Hospitals

BY JAMES MCKENTY, M.D.

Winnipeg

The hospital standardization program of the American College of Surgeons through its minimum standard during the past twenty years has effected a vast improvement in the conduct and equipment of hospitals upon this continent. The fact that when the first survey was made in 1918 only 12.9 per cent of the 692 hospitals examined were found to meet the requirements of the minimum standard is ample evidence that a reform was then much needed. In 1936 the survey of the same class of hospitals (100 beds and over) found 94.2 per cent worthy of approval. This reform initiated and carried out by the profession through the tactful agency of the American College of Surgeons is a great achievement, but two features of this program should, in the interests of the general practitioner especially, receive correction.

1. The minimum standard requires hospital directorates to judge of the professional competency and ethics of practitioners seeking permission to practise in hospitals. This judgment is made as a rule upon the advice of the advisory committee. Inasmuch as the license to practise of those excluded from hospital facilities is greatly reduced, a representative of the licensing body, the College of Physicians and Surgeons in each province, upon the advisory committee when it is dealing with such matters would give at least a semblance of regularity to its decisions.

2. A tendency to unduly magnify the responsibility of hospitals has been and is still found in the literature published by those members of the profession associated with hospital affairs. I quote from the *Bulletin of the American College of Surgeons*, January, 1924, "Hospital authorities do not have a divided responsibility for the care of patients. They are responsible for both the hospital care and the medical treatment." If the term "hospital authorities" be accepted in its legal meaning the above statement is, in Canada, not true, as is shown by the decision of the Supreme Court of Canada in the case of Nyberg *vs.* Provost Municipal Hospital Board. In that case the Chief Justice of the Supreme Court of Canada, in referring to the case of Hillyer *vs.* Governors of St. Bartholomew's Hospital, stated on page 229 of the Canada Law Reports for the year 1927, "That case is authority for the propositions, (*a*) That the relation of master and servant does not exist between an hospital Board and the surgeons and physicians whom it may employ for the treatment of patients in the hospital; (*b*) That the nurses on the staff of the hospital while they are actually engaged in assisting a surgeon during an operation are immediately subject to

his orders and control and that they are for the time being not to be regarded as servants of the hospital authority, and (*c*) That in regard to them while so engaged as in regard to the surgeon himself whom they are assisting, the only undertaking of the hospital authority is that they are qualified for the duties assigned them and not that they will not be negligent in their performance.'' The hospital corporation is, however, responsible for the work of its paid servants such as nurses and orderlies as has been shown by many successful damage suits for defective work on their part. Members of the professional staff are therefore neither servants nor agents of the governing body of the hospital.

In Canada the patients in the public or free wards, according to Miss Charlotte Whitton, constitute less than 40 per cent of the total hospital population. These are placed under the care of the honorary attending staff which is appointed by the hospital directorate on the advice of its advisory committee. The remaining 60 per cent choose their own doctor, and, upon his advice, also choose the hospital in which treatment is to be carried out. The doctor thus employed is expected by his patient to choose a properly equipped hospital. The hospital is, therefore, an agent through which the latest achievements of science are made available in the treatment of the sick. That is its main function, but the American College of Surgeons through its minimum standard has imposed on hospitals the duty of judging the professional competency and ethics of the members of its professional staff. This was a duty sorely needed but which the profession had neglected to perform. The imposition of this duty upon hospitals, along with the exaltation of the responsibility of hospital directorates, has tended to place hospitals in a dominating position and the profession in a subordinate position in the field of therapy.

Men and Books

WALTER HENRY, ARMY SURGEON

By WILLIAM BOYMAN HOWELL, M.D.

Montreal

Nearly a hundred years ago the autobiography of a British Army surgeon named Henry was published at Quebec.[1] Printers and publishers were scarce in Canada then, and the lapse of time, combined with the vagaries of collectors of Canadiana, have given the book a value in money never foreseen by its writer. But it has a value apart from that set on it by second-hand book-sellers; it is, unlike many of the early Canadian books, worth reading—though one has to forgive the writer much; for speaking of ''the cup that cheers, but not inebriates'', for calling fish ''the finny tribes'', trousers ''inexpressibles'', and eyes ''optics'', and his head his ''caput''. In one place drugs are ''Galenicals''. Not the least of his offences is a habit of breaking into apostrophes.

Walter Henry was an Irish Protestant. He was born in Donegal on New Year's Day, 1791. He had the best of educations, for he was brought up to love good books and good fishing. As a boy he spouted passages from Horace and Virgil, Milton and Shakespeare, while he cast for salmon on the banks of the river Esk. He devotes a whole chapter of his autobiography to his observations of the habits of salmon, and so completely does the subject absorb him that he forgets to be facetious and is merely sensible and interesting.

During Henry's boyhood the world was in arms. France was at war with her neighbours and striving to make them swallow draughts of republicanism, of that which had made her drunk. Ireland was in its normal state of unrest. Bands of Irishmen were roving the country in search of arms with which to compel other Irishmen to do something they did not want to do, or to prevent them from doing something they did want to do. But the laws were less calculated than they are now to protect the lawbreaker from the results of his actions. One night when Henry was a young boy a party of rebels in search of arms visited the Henry household. Walter was the first to hear them coming and aroused his father who armed himself with a blunderbuss, and his two men-servants with guns.

> A window was then cautiously opened, and a parley ensued; but as the rebel party, apparently ten or twelve in number, insisted on all the arms being given up incontinently, threatening death in case of refusal, and my father peremptorily declined the terms, the negotiations were soon concluded. The villains then prepared to force the back door, and one strong ruffian had already battered in two or three panels with a sledge hammer, when a window above his head was suddenly opened and my father shot him dead on the spot. Some desultory firing then took place; the gang uttered the most horrid threats of fire and extermination to all in the house; but another of them being hit, they became alarmed, and finally decamped, carrying with them the dead body.

When the time came for Henry to decide upon a profession he chose medicine. Curiously enough he makes no mention of any period of apprenticeship. He spent a winter in Edinburgh and then went to London where he studied surgery under Sir Everard Home and Sir Benjamin Brodie. It was an age of heroic medical treatment by blistering, sweating, purging, and most of all, by bleeding. The age of bleed-

ing had lasted since Hippocrates, and was nearing its end, but before the end came it was to reach the zenith of its popularity among medical men. Henry's readiness to open a vein at even the smallest excuse is shown all through his autobiography. His first act after obtaining his license from the Royal College of Surgeons was to apply for employment in the medical service of the army, in the hope of being sent to the Peninsula. Wellington was at that time holding the lines of Torres Vedras. Henry was accepted, gazetted "hospital mate", and a week later left London for Portsmouth where he was to embark for Portugal. He started forth to war, clad, not in sensible khaki but in a red coat and cocked hat. In the top of his cocked hat he wore the badge of the army doctor, a long black feather. He does not mention his trousers, but it is likely that they were of white duck, and starched at that.

He landed at Lisbon and was billeted at the house of a Portuguese gentleman, with whom, as neither knew the other's language, he was fortunately qualified to talk in Latin.

> Exactly opposite to Don Manoel's house there resided two good-looking Portuguese girls, who passed the greater part of their time, when the shade of the house or the cool of the evening permitted, in the handsome gilded balcony. I watched their manœuvres with a good deal of attention, and found that their principal amusement consisted in playing tricks on the Gallegos, or Gallician water-carriers, passing beneath, with their little painted barrels on their shoulders. No sooner did a Gallego approach than these damsels would accost him 'tio! tio!' 'Uncle-Uncle', the familiar term used to the lower classes. The man would stop and look up; then—I shudder whilst I relate a deed so unlady-like and atrocious—the playful girls would giggle and spit in his eye. The water-carrier would retort by a squirt from the pipe of his barrel about the ladies' ankles, and this was considered great fun on both sides.

Henry's experiences in the Peninsular War were in many respects very similar to those of the average young medical officer who served with the British Army in France and Belgium during the Great War. There were long periods when he had little to do; there were short periods when he had much to do. He had plenty of time to spend in sightseeing, in not very strenuous attempts to learn the language of the country, and in going to social gatherings at which he and his young fellow-officers ate and drank quite as much as was good for them. There was boating sometimes, and fishing. He made love to Portuguese and Spanish ladies when opportunity arose. The course of his love affairs was not always smooth. Once, when he was whispering soft nothings into the ear of a temporary sweetheart, her aunt suddenly appeared, tore her from his arms, and dragged her off to an accompaniment of what Henry describes as "terrific slaps". Another of his love affairs contained some of the ingredients of a melodrama — an anonymous letter, a plot to assassinate him, and the foiling of a Spanish rival. In the last act the stage was set for an elopement, but Marshal Soult chose this time to make a rapid advance, and the British fell back. Henry had to leave at short notice. He might still have carried out his scheme, but the donkey upon which his bride was to have ridden to the church went lame. If we may judge by one of Henry's experiences, the P.M.O. of the Peninsular War was less strictly conventional than the A.D.M.S. of a hundred years later; though similar to him in manner.

> When I first landed I had called on Dr. B—, the principal medical officer . . . who received me as graciously as his nature permitted. After a short conversation, in the course of which he directed me to call the next morning for further instructions, and when I had risen from the chair, preparatory to taking my leave, we were both startled by a shrill, violent, and prolonged female shriek from the upper part of the house. The principal medical officer suddenly turned as pale as a sheet, and exclaiming, 'By G—, she has killed herself!', rushed out of his office and ran up the stairs.

The alarm turned out to be nothing more serious than an attempt on the part of the principal medical officer's mistress to commit suicide by inflicting a superficial wound on herself with a stiletto. Henry was summoned upstairs by his agitated senior officer to staunch the bleeding and dress the wound.

After some six months spent in performing various kinds of duty in various parts of Portugal, Henry was, in December, 1811, appointed assistant surgeon to the 66th Foot, the Bedfordshire Regiment of today. A month later he started off on horseback from Lisbon to join his regiment, going first north to Coimbra and then southeast to Elvas. After travelling five hundred miles and spending more than two months on the journey, he found the 66th at Merida, a town on the Guadiana, forty miles above Badajos. His way had taken him past Badajos which was being besieged, and from Albuquerque, twenty miles off, he had seen the smoke and heard the growling of the guns. The town fell a few days later when he was in the neighbourhood again, this time in charge of a convoy of sick, and he rode into the town to see the sights.

> I reached the bridge over the Guadiana in three-quarters of an hour, but my surprise was great; instead of finding everything quiet and everybody occupied in attentions to the wounded, and preparations for burying the dead, as I had expected, I beheld a scene of the most dreadful drunkenness, violence and confusion. Parties of intoxicated men, loosed from all discipline and restraint, and impelled by their own evil passions, were roaming and reeling about; firing into the windows, bursting open the doors by the discharge of several muskets simultaneously against the lock—plundering—shooting any person who opposed them—violating, and committing every kind of horrid excess, and sometimes destroying each other . . .
>
> I proceeded amidst desultory but dangerous firing, by the *detour* of the Talavera gate to the main breach . . . There lay a frightful heap of fifteen hundred British soldiers, dead but yet warm. and mingled with

some still living, but so desperately wounded as to be irremovable without more assistance than could yet be afforded—there they lay stiffening in their gore—body piled upon body—involved, intertwined, crushed, burned and blackened—one hideous and enormous mass of carnage.

Malaria was common in Spain during the summer, and Henry had a severe attack. He was looked after by a colleague who lacked faith in the orthodox treatment by copious and repeated bleedings from the arm and from the temporal arteries. Nevertheless when symptoms of "determination to the head" had become evident, Henry's hair was shaved off and three dozen leeches applied to his scalp; he was then carried into the courtyard of the house, where, having first placed him in an erect position, his friends poured a couple of dozen bucketfuls of cold water on his head from a third storey window.

For the first two hours I was not sure whether my head had not been carried away in the flood, for I felt as if there was no living part, and all was numb and cold above my shoulders—but there was violent reaction during the night and I became delirious the next morning.

His recovery from the malaria and from his friends' well-meant efforts to cure him was due to the care he received from a Portuguese priest upon whom he was billeted.

When he recovered he started off to find the 66th, and on the way had a relapse. He was without money, for the pay of the army at that time was six months in arrears. However, he had a kind-hearted commanding officer who sent him three doubloons borrowed, Henry says, from a Turkish sutler. Shortly after he rejoined his regiment, the brigade to which it belonged received its marching orders. It was midsummer, the country was parched, and the roads deep in dust. One evening, after a particularly hard day, the brigade halted near a village fountain. The soldiers threw themselves down beside it and drank their fill; many of them dipped their heads in the cool water. Next morning nearly 150 men of the brigade reported to the regimental surgeons that they were spitting up blood. This trouble was found to be due to the presence of leeches which had been taken in with the water at the fountain, and had attached themselves to the mucous membrane of mouth, nostrils, pharynx, and gullet. Various measures were used to dislodge them, but none was very sure. It was not enough to tear away the body since the head was usually left attached to the mucous membrane. Strong emetics were used to remove those that were believed to have gained access to the stomach.

At the Battle of Vittoria Henry acted as assistant to the staff surgeon of the second division. Soon after the fighting began he was sent off to help the regimental surgeon of a brigade which had sustained heavy losses.

We collected the wounded in a little hollow, out of direct line of fire, but within half musket shot—unpacked our paniers and proceeded to our work . . . This brigade had, I believe, between four and five hundred men put *hors de combat* in the course of an hour; so we were fully employed. A stray cannon shot from a distant battery would drop among us occasionally, by way of a hint to inculcate expeditious surgery. After one of these unpleasant visitors had made its appearance, a young chirurgien of my acquaintance, who is still living, became so nervous that although half through his amputation of a poor fellow's thigh, he dropped his knife and I was obliged to finish. At my suggestion he lay down on the grass, took a little brandy, and soon recovered and did good service the whole day.

One shudders to think of the results of operations performed in such an environment. There were no anæsthetics and no antiseptics; competent help was scarce and equipment primitive; and there was desperate need of haste. Probably few of the wounded recovered from an amputation. There was a frightful mortality, indeed, after all sorts of wounds. Yet there were astonishing recoveries too. Henry mentions an officer, a friend of his, who was wounded in the lung and saved by "judicious bleeding"; the amount of blood withdrawn amounted to three hundred ounces in two days! Before the day was over Henry was sent to help with the wounded of another brigade. By the end of the afternoon the French were retreating, but not through the pass by which they had hoped to reach France in case of a defeat. The road from Vittoria to Bayonne had been seized by the British, and the retreat became a rout. The wealth wrung from the Spanish was abandoned. Alison, in his "History of Europe" says:

No estimate can be formed of the amount of private plunder which was taken on the field, but it exceeded anything witnessed in modern war; for it was not the produce of the sack of a city or the devastation of a province, but the accumulated plunder of a kingdom during five years, joined to the arrears of pay from the invaders' host for two, which was now at one fell swoop reft from the spoiler. Independent of private booty, no less than five millions and a half in the military chest of the army was taken, and of private wealth the amount was so prodigious that for miles together the pursuers may be said to have marched upon gold and silver without stopping to pick it up . . . Wives and concubines, nuns and actresses, arrayed in the highest luxury of fashion, were taken by hundreds. Rich vestures of all sorts; velvets and silk brocades, gold and silver plate, noble pictures, jewels, lace, cases of claret and champagne, poodles, parrots, monkeys, and trinkets, lay scattered about the fields in endless confusion, amidst weeping mothers, wailing infants, and all the unutterable miseries of military overthrow.

During the battle Henry went about his duties encumbered with a heavy bag of gold entrusted to him by an officer who in the morning had had a presentiment of death. The sequel of the incident was unusual in the history of presentiments before battle; the officer was not killed. After Vittoria there was plenty of hard fighting before the French were driven across the Pyrenees and the British came in sight of the

plains of Gascony. Soult re-organized the French army and attacked the allies who had to fall back. It was late in the autumn before Wellington had sufficient forces at his disposal to drive the French finally back into France.

At this point in the autobiography we catch a glimpse of Wellington through Henry's eyes. The church at the village of Maya had been turned into a dressing station, and Lord Wellington stopped on his way through the town to ask after the wounded.

> I went out, and for the first time, had the honor of a short conversation with his Lordship. He was pleased that so few men had been hit, and that their wounds were, for the most part, slight. He rode on, saying courteously, 'Good-morning—go on with your duties.'

Henry was present at the battle of the Nivelle. His patients were not exclusively military on that strenuous day. A young French lady looking from a window at the fighting had her elbow shattered by a grape shot, and in the evening Henry amputated her arm. The 66th took part in the fighting around Bayonne.

> I had good professional practice on the 13th, for we (the British) had twelve or thirteen thousand wounded. Some wounds were frightful, yet the patients recovered, and I may mention, amongst the worst, the case of Captain Bulstrode of the 66th regiment, who received a grape shot in the lower jaw—the ball carrying away one side of the jaw entirely, and burying six teeth in the tongue, which I extracted at the time. The officer recovered and I had the pleasure of dining with him afterwards in London.

It was the custom at the time of the Peninsular War for a certain number of soldiers of every regiment to bring their wives with them when on active service. These women, riding on donkeys or walking, accompanied the regiments on the march, foraged for their husbands and when fighting occurred, looked on from some point of vantage. One of them was knocked over by a cannon ball.

> I saw her immediately, but found there was only a graze, and slight contusion on the shoulder. At first she was sadly frightened, but when I assured her there was no harm done, she was so delighted that she pulled a fowl out of one enormous pocket and half a yard of black pudding out of the other, of which she begged my acceptance.

There were many casualties at the storming of the hill of Carris, near St. Palais. Henry was the first surgeon to reach the summit and was in great demand. He tells the following gruesome story of what befell after the fight.

> Fifty or sixty unfortunate wretches concealed themselves in a house at the top until the affair was over, and our men had piled their arms and were kindling night fires; when they sallied out in a body and attempted to escape down the hill. At the first rush our soldiers seized their arms, which were loaded—pursued them with a loud cheer, and shot or knocked down almost every man. They were stripped soon after—for this process takes place in a very short time—and I recollect when the moon rose seeing their plump white corpses scattered over the field. In the morning we were all shocked to see their bodies mutilated of their fair proportions, and all the fleshy protuberant parts cut clear off down to the bone. How this happened none could say; although a report was current at the time that a certain ingenious regiment of Byng's brigade had metamorphosed the poor defuncts' hams into pork, and exchanged them with the Portugese troops for rum. One of our officers averred that he heard two of the culprits chuckling at the trick.

Henry had more than one opportunity to observe Wellington's sharp method of dealing with looters in France. One day he and another officer came to a farm house where they found an old man who had been beaten and robbed by some soldiers. The matter was reported to headquarters. One of the assailants had been wounded in the thigh by the farmer; he was caught, identified and summarily hanged in the presence of Henry's division.

> After forty-five minutes I was ordered to ascertain whether life was extinct, that the men might not be kept longer in the cold than was necessary.

In April, 1814, when the 66th was marching into Toulouse, news came of Napoleon's abdication, and peace was declared. Napoleon's popularity in France had been on the wane for some time as a result of the reverses sustained by his armies, and a strong feeling in favour of the Bourbons had developed. In the southwest the ruthless exactions of the French soldiers from their own countrymen had caused widespread suffering and exasperation. Wellington, on the other hand, after he crossed the Pyrenees and entered France, conciliated the inhabitants in the districts in which his army was operating, by forbidding all plundering and by paying for everything that he requisitioned. The British were looked upon as deliverers of the people from the yoke of Napoleon, and after his abdication were treated with the greatest friendliness. There were six pleasant weeks at Pourville, near Toulouse, and then in June the regiment started on its march to Bordeaux.

> Our route lay down the left bank of the Garonne, one of the most rich and lovely tracts in France. The marches were short — the inhabitants overwhelmingly civil and we had a ball every night.

On the way Henry's faithful Portuguese servant left him to return to his own country. Henry celebrates the event with one of his most florid apostrophes.

> And there too, excellent and faithful Antonio! There didst thou separate from thy master and wend thy way to thy native Coimbra, and to astonish the untravelled simpleness of Mondeg by the relation of thy various adventures, &c, &c.

At Bordeaux the 66th had three weeks before embarking for England. Henry, billeted in a château with seven other officers, found the time pass rapidly enough. They had plenty of money to spend, for arrears were being made up at the rate of two months per week. Claret was abundant and good; and whatever virtues the

eight officers practised temperance was not among them.

On his return to England Henry obtained leave of absence and spent three months at his home in Ireland. He rejoined the 66th at Newport in the Isle of Wight, and in January, 1815, marched to Chatham with a detachment of eight officers and a hundred men who were being sent to India to re-enforce the first battalion of the regiment. The troopship, an East Indiaman, lay rolling at anchor in the Downs for more than a month, prevented from proceeding on her way by calms and baffling winds. The monotony of life on board was broken suddenly in the month of March by the news that Napoleon had escaped from Elba and was once more in France. The voyage to Madras lasted more than three and a half months. At Calcutta Henry saw the adjutant bird for the first time.

> The adjutant is a harmless and useful bird, that performs the duty of scavenger in India—devouring offal, and punishing snakes, of which he is very fond. His valuable services are so appreciated that the Company have taken him into their charge and placed his whole fraternity under their protection—punishing with a heavy fine the murder of one of these birds. Yet such is the ingratitude of mankind that the poor inoffensive adjutant is persecuted by the most annoying and cruel tricks. Shank bones of mutton are cleaned out and stuffed with gunpowder, with a slow match applied—then the meat is thrown and swallowed, and when the poor wretch is chuckling over his savoury morsel it explodes and blows him to atoms.

From Calcutta Henry went by boat up the Ganges and the Hoogly, having much good duck and snipe shooting on the way, and arrived at Dinapore in time to accompany his regiment on a march to Nepaul, for there was war with the Ghurkahs. Henry saw no fighting as peace was declared before the 66th came up with the enemy.

During the expedition one of his fellow-officers suddenly went out of his mind as the result of hard drinking.

> He was sitting up in bed, with his writing desk on his knees, hard at work, his face was much flushed—eyes bloodshot — appearance altogether very wild, and two pistols lying on a camp table beside him. At the first glance I saw that mischief was impending, but said nothing more than the usual salutation and sat down. He continued his writing for a quarter of an hour, then addressed me, 'Doctor, I am making my will —I have left you my books and my sabre, as mementoes of friendship. Do you see those pistols? Examine them —well, you find they are loaded—one is destined for Colonel Nicol, (this was the commanding officer of the 66th), a ball from the other will finish my career.'

Henry dealt with the situation in a manner as simple as it was effectual. He bled the officer until he fainted, then secured his pistols, sword, and razors; after which he put him under restraint. Not long afterwards this same officer, after drinking a bottle of brandy, drew his sword, and rushed about threatening the lives of his native servants. He wound up this escapade by jumping into the Ganges and drowning himself.

The campaign against the Ghurkahs over, the 66th spent three months at Dinapore, and were then ordered to proceed in boats to Cawnpore. On the way Henry amused himself by taking pot-shots at the vultures which were feeding on the putrefying bodies floating down the Ganges. At one place he saw suttee performed. The widow was a girl of seventeen. He and some of his fellow-officers tried to dissuade her from sacrificing herself, but ineffectually. Her cries when the flames reached her were rendered inaudible by a "horrible din of tom toms, gongs and human voices". There were no barracks ready for the 66th when they reached Cawnpore, so for three weeks they stayed on board their boats. Remittent fever broke out, and in the course of the next four months carried off five officers and 150 men. Only about half a dozen men escaped an attack. As usual, Henry found bleeding to be the best treatment. After six months in Cawnpore the 66th received orders to return to Calcutta whence it was to sail for St. Helena to form part of the garrison guarding Napoleon. Henry embarked on April 2, 1817, with the headquarters of his battalion and three hundred men; and after a voyage of a little more than three months reached St. Helena. Soon after landing he was sent with part of his regiment to Deadwood Barracks, half a mile from Longwood.

Napoleon was the subject of interminable discussion in the messes of the 66th; his comings and goings, his health, his bickerings with Sir Hudson Lowe, and the doings of the members of his household. It was not until three months after their arrival, that, through the good offices of Sir George Bingham, they were allowed to go to Longwood to be presented to the Emperor. The interview took place in an antechamber.

> We entered, formed a ring around the room, according to seniority, and in about a minute Napoleon walked into the circle. He was dressed in a plain dark green uniform coat, without epaulettes or anything equivalent, but with a large star on his breast, which had an eagle in the centre . . . He had on white breeches with silk stockings, and oval gold buckles on his shoes—with a small opera hat under his arm. Napoleon's first appearance was far from imposing—the stature was short and thick—head sunk into his shoulders—his face fat, with large folds under the chin—limbs appeared to be stout, but well-proportioned—complexion olive—expression sinister and rather scowling.

Napoleon walked round the room speaking to each officer in turn. To one not accustomed to mixing with emperors some of his questions might seem impertinent. For instance, he asked Col. Nicol, the commanding officer, whether his officers got drunk, and a Captain L'Estrange why his complexion was so dark. Henry, when his turn came, was questioned on the subject

of bleeding in the treatment of disease, and he admitted that he had great faith in it, which indeed he certainly had.

Barry Edward O'Meara, formerly surgeon of the *Bellephoron* was living at Longwood at this time in the capacity of physician to Napoleon. He sent for Henry one day to consult him about Cipriani, Napoleon's *maître d'hotel,* who had been taken ill with "inflammation of the bowels" (probably appendicitis). Henry's attendance continued until Cipriani died of "mortification of the bowels" (probably general peritonitis). Cipriani had served Napoleon faithfully for more than twenty years; his room was under the same roof; yet during his last illness Napoleon never went to see him.

> Some time after Cipriani's death, Mr. O'Meara called on me at Deadwood, with a smiling countenance, to tell me he was the bearer of good news, on which he offered me his congratulations. The Emperor had consulted him as to the propriety of giving a fee as a present to the English Physician, who had attended his servant; and the result was that a present had been preferred. Mr. O'Meara added that Napoleon had condescended to enquire the name of the English Doctor, and whether he was married or single; and that the business had ended in an order having been given for a Breakfast Service of Plate, to be sent for to Rundell and Bridges, Fleet Street, London.

There was a strict rule at St. Helena that no British officer might accept a present from Napoleon or from any of his staff and Henry declined the silver breakfast service. He was then asked if he would accept it clandestinely. Realizing that to do so would be not only to disobey orders but to put himself in the power of the Bonapartists, he very wisely refused.

The position of physician to the Emperor was beset with pitfalls. O'Meara became *l'homme de l'Empereur* and was dismissed from the King's service by court martial for alleging, without a particle of evidence, that Sir Hudson Lowe had tried to suborn him to poison Napoleon. He afterwards published a book entitled *Napoleon in Exile; or A Voice from St. Helena,* partly with the object of arousing sympathy for Napoleon in Europe, and partly to vent his spite against Sir Hudson Lowe and other officials at St. Helena. The book had a large circulation and brought him to the notice of a rich widow whom he married and with whom, no doubt, he lived happily ever afterwards. He was succeeded by a Mr. Stoker, surgeon of H.M.S. *Conqueror,* whose appointment also ended in a court martial and dismissal from the army. Dr. Verling, of the Royal Artillery, was next appointed and was offered a salary of 12,000 francs as the Emperor's private physician, provided of course he would be the "Emperor's man". He refused the bait and, though he lived at Longwood for eighteen months as Napoleon's physician, was never allowed to see him. Finally, the Bonaparte family sent out a Corsican doctor named Antommarchi, who was an excellent anatomist and pathologist but an indifferent clinician. He remained at Longwood until Napoleon's death.

Henry became a frequent visitor at the house of Marshal Bertrand. One day he found Mme. Bertrand much upset because Napoleon had shot two pet kids that belonged to her children and had strayed into the Emperor's Chinese garden. According to Forsyth, the Emperor enjoyed the sensation of shooting the pet kids so much that he sent a servant to Sandy Bay to buy more kids for him, to be used for the same purpose. Among the many other innocent victims of the Emperor's lust for blood were a tame rabbit, three chickens and an ox; all trespassers in his garden.

It was proclaimed by the adherents of Napoleon that the climate of St. Helena was unhealthy and was the cause of the liver disease which O'Meara and Antommarchi believed him to be suffering from during his last illness. Emil Ludwig, in his "Life of Napoleon", makes his reader's flesh creep when he describes St. Helena; a horrible place with a deadly climate, situated in sunless tropics; the inhabitants, to a man or woman, suffering from liver disease; the British sailors dying by hundreds unless the ships were kept out at sea, under sail; and, worst of all, Longwood, a pestiferous tableland chosen as a suitable place to make a sick foe die without any undue delay. Henry, qualified to speak with authority of the climate by four years' residence during which he was medical officer of a regiment of twelve hundred men, gives a different account.

> For a tropical climate, only fifteen degrees from the Line, St. Helena is certainly a healthy island—if not the most healthy of this description in the world. During one period of twelve months, we did not lose one man by disease, out of five hundred of the 66th, quartered at Deadwood. In 1817-'18-'19, the thermometer at the hospital there ranged from fifty to seventy-five degrees, Fahrenheit, with the exception of two calm days, when it rose to eighty degrees. The upper parts (of the island) were decidedly the most healthy; and we often moved our regimental convalescents from James Town to Deadwood for cooler and better air.

Perhaps the discrepancy of these two accounts may be explained by the fact that Henry was not writing a "best seller". The real reasons why the British men-o'-war were kept at sea were: firstly, because they had to patrol the waters around the island on the look-out for any ships approaching with the purpose of rescuing Napoleon, and, secondly, that there was no harbour and they were liable to be caught on a lee shore if they remained at anchor off James Town.

Napoleon died on May 5, 1821. An autopsy was performed by Antommarchi in the presence of all the medical officers of the garrison and of some of the fleet, of Marshal Bertrand and Count Montholon and some officers of Sir Hudson Lowe's staff. The cause of death was found to

be cancer of the pylorus. Even Antommarchi had to admit this. In his report he said.

> The Liver, which was affected with chronic hepatitis, was closely united by its convex surface to the diaphragm . . . The stomach appeared at first to be in a most healthy state, with no trace of irritation or phlogosis, and the peritoneal membrane presented the most satisfactory appearance. But on examining this organ with care, I discovered on the anterior surface, towards the smaller curve and three fingers from the pylorus, a slight obstruction of a scirrhous nature, of very small extent and exactly circumscribed. The stomach was pierced through and through in the centre of this small indentation.

This description differs considerably from that contained in the official British report which states:

> . . . the internal surface of the stomach to nearly its whole extent was a mass of cancerous disease or scirrhous portions advancing to cancer; this was particularly noticed near the pylorus.

The official report, drawn up after the autopsy, was based on Henry's notes. Antommarchi was asked to sign it and was on the point of doing so when he was called aside by Marshal Bertrand and Count Montholon. After a conference with them he refused to sign. It would not have been expedient to acknowledge that the diagnosis which had been used to bring odium on the British government was wrong. Henry, being a junior officer, was not asked to sign the report. Two years after Napoleon died Henry wrote a letter to Sir Hudson Lowe which he does not mention in his autobiography. In it he made this statement about what occurred at the post-mortem on Napoleon.

> When the liver was next examined the countenances of the spectators indicated much anxiety. When M. Antommarchi made his first incision into it (the liver) he expected to see a flow of pus from the abscess which had been anticipated in its substance, but no abscesses, no hardness, no enlargement, no inflammation were observed. On the contrary, the liver was of natural size and perfectly healthy in internal parts.

Henry's first impression of Sir Hudson Lowe was somewhat unfavourable, but later he came to like and admire him.

> I firmly believe that the talent he exerted in unravelling the intricate plotting constantly going on at Longwood, and the firmness in tearing it to pieces, with the unceasing vigilance he displayed in the discharge of his arduous duties, made him more enemies than any hastiness of temper, uncourteousness of demeanour, and severity in his measures, of which the world believed him guilty.

One night, not long before he left St. Helena, Henry was sent for in great haste to go to Government House. Sir Hudson Lowe's youngest child had been taken suddenly ill.

> I found the little patient—an infant of eight months—apparently gasping its last under a terrible attack of croup; and the peculiar distressing sound of the spasmodic breathing audible over half the house. It was plain that without prompt relief the poor child would be lost. 'The child must instantly be bled', I said. 'Good God! Sir, bleed an infant of this age!' 'Yes', was my reply, 'else the child will be dead in ten minutes'. 'But doctor, you won't be able to find a vein'. 'We'll try'. So the little sufferer's arm was bandaged, and when three ounces of blood had flowed, the breathing became comparatively quiet and easy; and after some medicine had been given, the child fell into a sound sleep.

There was no need for a strong garrison at St. Helena after Napoleon died, and the 66th Foot received orders to prepare for embarkation. Henry sailed in a 500 ton ship, the *Camel,* with the band and two companies of his regiment. Napoleon's suite sailed in the same vessel: Marshal and the Countess Bertrand and their children, Count Montholon, Dr. Antommarchi, the Abbé Vignali and some servants. Pent up in a little vessel during the two and a half months of the voyage, Henry and his fellow-officers came to know their French fellow-passengers intimately, but never heard them utter any complaint against Sir Hudson Lowe. Count Montholon is reported to have once said to a British officer, "Mon cher ami, an angel from Heaven could not have pleased us as governor of St. Helena".

Shortly after his return to England Henry got leave of absence for six months to attend the hospitals of London and Paris. In March, 1823, the 66th received orders to march to Liverpool.

> The weather was very cold and inclement at this time and in the course of one long and circuitous march . . . the men were up to their knees in snow and sleet half the way. In consequence, almost every man caught a severe cold; and many contracted bad chest inflammations; dangerous at the time, and in some instances productive of evil consequences in the shape of consequent consumptive complaints . . . When we reached Leeds the whole regiment began to cough, as if from one common impulse; and on examination the great majority were found feverish; with pain in the chest and impeded respiration. The writer collected all the sick in the large yard of the Inn where the officers messed, and having opened a dozen veins at once, he bled them nearly to a man.

That so many became ill as a result of this wetting is explained by the physical condition of the British soldier at the time. His health was invariably below par. The sanitary conditions under which he lived were deplorable. He was housed in barracks which were little better than dog kennels; he was badly clothed and under-fed; and he made up the deficiencies of his diet by hard drinking. The mortality among soldiers in the British Isles was 15 per thousand; an appalling figure for young men chosen at their enlistment for their good physique and freedom from disease.[2]

At Liverpool the 66th embarked in "some miserable schooners" and sailed for Dublin. The next three years were spent in different parts of Ireland. Wherever the regiment was quartered there seems to have been the best of hunting, shooting, and fishing for Henry and his brother officers, besides social entertain-

ments of every description. During his time in Ireland, Henry was promoted from assistant surgeon to surgeon, his senior officer having died of what seems to have been typhoid.

In June 1827 the 66th sailed from Cove on board H.M.S. *Romney* and the transport *Arab* for Canada. Henry had heard much of the beauties of Quebec but was disappointed.

> We found the town an ugly cluster of houses, pitched on the extremity of a bald promontory—the streets narrow and crooked, and those built on made ground round the base, disgracefully filthy—with zigzag wharves pushing irregularly into the noble stream, whose surface was dotted with shipping.

However, on further acquaintance, the beauty of the views from the citadel and in the neighbourhood, combined with the historical associations, compensated him to a certain extent for having to live in such a grubby little town. After spending three years in Quebec and a year in Montreal, the 66th went to Kingston, Ontario. At each of these places Henry managed to get excellent fishing. There were salmon and trout near Quebec. He speaks of the enormous number of shad in the St. Lawrence near Montreal and in the Ottawa. Large bass were to be caught with the greatest ease in the waters of Lake Ontario around Garden Island and in the Bay of Quinté.

Malaria was common in many parts of Canada at the time, and during their first summer at Kingston many of the personnel of the 66th went down with it. During the years 1831-32 Asiatic cholera was epidemic in Europe. Most medical men in Canada believed that the disease could not cross the Atlantic. They were wrong. It broke out in Quebec on June 8, 1832, and three days later in Montreal. The first case in Kingston, a fatal one, appeared on June 17th. There was great activity in the barracks; much lightening of fatigues; and daily inspections by Henry and his assistant, both of them, one may be sure, having an eagle eye for an excuse to bleed.

> Although the cholera raged in the town for the next fortnight, we had no case in the regiment till July 4, when two grenadiers were attacked with frightful spasms. I was sent for on the instant, bled them both largely, and they recovered. Ten other men of the regiment were taken ill and treated in the same way; the agonizing cramps yielded to the early and copious bleeding as to a charm, and they recovered.
>
> Encouraged by the result of these, and several other instances amongst the poor people of the town, I began vaguely to imagine that this plan of treatment would be generally successful, and wrote confidently to this effect to Dr. Skey; but I was soon to be undeceived. Three men and a woman of the 66th were attacked the same night. I saw them immediately; and the symptoms being the same to all appearance, they were bled like the others, and died within twelve hours of the first attack.
>
> We all heard wonderful accounts of the effects of transfusion of saline fluid* into the veins, and Dr. Sampson, the principal practitioner in Kingston, and a man of talent, was determined, as well as myself, to give it a fair trial. We used it in twenty bad cases, but unsuccessfully in all . . . We had thirty-six cases of bad cholera, besides a host of choleroid complaints in the regiment. Of these we lost five men and two women. No child suffered.

* The saline fluid used was approximately of the same strength as the "normal saline" of today.

After spending a year at Kingston, the 66th was sent to York, afterwards Toronto. Henry describes York as a "long, straggling place, recently redeemed from the forest, running two miles along the lake". The population, he says, was highly moral and respectable. They are still respectable. There was good snipe shooting in the outskirts of the village. For three or four days every spring, wood pigeon, migrating north, used to cross the upper end of the lake in prodigious numbers, and reach land near the barracks where they could be knocked down with sticks. Salmon were caught in nets or speared in Lake Ontario at this time.

In May the 66th was returned to Kingston. Two months later there was an outbreak of cholera, even more severe than that of 1832. Of the 769 officers and men, with their wives and children who were under Henry's care in the 66th, only eight caught the disease. Of these, three died. The mortality in Kingston itself was between 16 and 17 per cent of the population.

> The writer's favourite remedy was castor oil combined with a small quantity of laudanum, given in some grateful and demulcent fluid, as hot as possible—making the patient lie on the right side for the assistance of gravitation towards the pylorus. In some hundred cases on this and the former occasion, he witnessed the most excellent effects from this remedy; and moreover, experienced them himself in the early stage of attacks he had at Kingston. Once, when attending a gentleman who had died of this disease, the writer was conscious of the very moment when he contracted it by the patient's bedside—instantly went home and to bed, and took the oil and laudanum—when five minutes' delay might have cost him his life. For some time there was a terrible internal conflict—the heart and whole system laboured tumultously, and the balance appeared to vibrate between the fatal rush of serum, or thin fluid of the blood, to the coats of the intestines, and a salutary determination to the exterior. All this time the pulse could not be counted and the feeling of anxiety and oppression was dreadful. At length the circulation became calmer; the shrivelling skin swelled out with grateful heat and warm moisture; and the crisis was past. Here, and in many similar instances, like oil on a stormy sea, this invaluable medicine soothed the internal commotion and effected a calm.

From Kingston the 66th went back to Quebec. The political situation in Lower Canada had become steadily worse. Papineau and his followers in the House of Assembly had been a thorn in the side of many successive governors-general. When Lord Gosford came as governor he took special pains to entertain the politicians at Government House with a view to keeping them in good humour.

. . . some of the weaker members of the Assembly, fuddled a little by the unwonted good cheer, went occasionally to laughable extremes. One of them who chanced to sit next to His Excellency one day at dinner, vented his warmth of feeling towards his host by a fervent slap on the back accompanied by the compliment —'Milord, vous êtes bien aimable'. Lord Gosford's reply was, 'Pardonnez, c'est le vin'.

Henry ends his autobiography with seven pages of apostrophe to the Canadians. He pleads with the French Canadian to abjure Papineau and all his works and the Upper Canadian to turn his gaze away from the will-o'-the-wisp of responsible government. Fortunately, no one heeded him, and our present enlightened form of government was evolved—of government of the people, by the party, for the party.

Henry published his autobiography in 1839, the year in which he became staff surgeon. In 1845 he was appointed deputy inspector-general of hospitals, and in 1850, inspector-general of hospitals in Canada. In the same year he retired on half-pay, after having served forty-five years in the army. He died at Belleville, Ontario, in 1860.

REFERENCES

1. Trifles from My Portfolio, or Recollections of Scenes and Small Adventures during Twenty-Nine Years of Military Service in the Peninsular War and Invasions of France, the East Indies, Campaign in Nepaul, St. Helena and during the Detention and Until the Death of Napoleon, and Upper and Lower Canada. By a Staff Surgeon. Quebec, Neilson, 1839.
2. FORTESCUE, J. W.: History of the British Army, Macmillan, London, 1923, xi; bk. xxi, chap. i.

Association Notes

The Annual Meeting

It will be a matter of the greatest satisfaction to the whole medical profession that the conjoint sessions of the Canadian Medical Association and the Ontario Medical Association, to be held in Ottawa during the week of June 21st, next, will be graced by the presence of their Patron-in-Chief, His Excellency the Governor-General.

In accepting the invitation extended him, His Excellency has been pleased to intimate that it is his intention to be present as guest of honour at the luncheon on June 23rd, and that he will participate in the ceremonial function in the evening of the same day.

The Housing Committee recommend that all those who intend to visit Ottawa during convention week should refer to the hotel accommodation schedule which appeared in the February number of the *Journal,* and book their reservations without delay. Already a great many requests for accommodation have come in, and Dr. J. H. Alford, Chairman of the Local Committee, 235 O'Connor St., Ottawa, strongly urges that none of those who expect to attend the meetings should postpone making arrangements in this connection.

It will soon be possible to publish in full, details of the scientific program. To date 90 per cent of all those invited to take part have accepted. The following list of speakers for the General Sessions however is announced.

GENERAL SESSIONS

DR. A. T. BAZIN, Montreal;

DR. WM. MAGNER, Toronto;

DR. A. M. SNELL, Rochester, Minn.,
Symposium on Jaundice.

British Speaker (name to be announced later).

DR. A. H. GORDON, Montreal,
Medical Clinic.

DR. HERMANN ROBERTSON, Victoria,
Presidential Address.

DR. B. P. WATSON, New York,
Post-partum Sepsis.

DR. R. R. GRAHAM, Toronto,
Surgical Clinic.

DR. G. M. GELDERT, Ottawa,
Demonstration of the electrical stethoscope.

British Speaker (name to be announced later).

DR. H. B. CUSHING, Montreal,
The Blackader Oration.

SECTION ON SCIENTIFIC EXHIBITS

One of the important features of the Convention will be the section on scientific exhibits. This is a new venture for the Canadian Medical Association, the success of the scientific section at the 1936 meeting of the Ontario Medical Association in London having set a precedent. The Exhibit will occupy the entire mezzanine floor of the Chateau Laurier, and will be representative of every branch in medicine. It is the object of the Committee in charge to make this inaugural exhibit an outstanding success, in order that still greater benefits will be derived from attendance at the Convention.

This new feature of the combined sessions is already attracting a great deal of attention from all over Canada, and the Committee has received many requests for space in the exhibit.

It is strongly advised that all those who are interested in providing exhibits should communicate immediately with Dr. George S. Williamson, 295 MacLaren St., Ottawa, Chairman, Committee on Scientific Exhibits. Application

for space in the exhibit must be made upon official forms which can be obtained along with all necessary information from the Chairman.

COMMERCIAL EXHIBITS

As to the rooms allotted for the purpose of commercial exhibits, it is noteworthy that by the end of January more space had been sold than had been applied for at any previous medical convention in Canada during the past seven years.

MEMBERSHIP

It is the object of the Ottawa Medico-Chirurgical Society to enlarge its membership in the two major organizations by enlisting, as nearly as possible, all practitioners in Ottawa and vicinity. With this in view an organization meeting was held on January 30th at the Royal Ottawa Golf Club, where committee men to the number of 70 were entertained at dinner as guests of the President-elect of the Ontario Medical Association, Dr. R. K. Paterson. The enthusiasm which prevails locally bids fair to ensure the highest efficiency in the conducting of the Convention.

Hospital Service Department Notes

Hospital Planning in Sweden

Sweden is much to the fore these days with its efforts to promote social stability and welfare, and it is now giving us leadership in hospital planning as well as in economics. Sweden has been divided into "hospital districts" — 25 provincial districts and 6 large towns, 31 in all. In 1929 the Swedish Government appointed a committee to draw up a hospital plan for the country, this plan being submitted in 1934. At the same time a special study was made by the city of Stockholm of its local needs, and this has been described by Mr. Hjalmar Cederström, city architect of Stockholm in *The Hospital*. The principles underlying the solution adopted are worthy of consideration.

It was decided that special diagnostic and therapeutic facilities should be assembled in one or more central hospitals and not scattered in small special hospitals. This principle has been the deciding factor in modernizing, enlarging or discarding old hospitals. Three new central hospitals, one in each borough, are to be erected. One of these, of 1,500-beds capacity, is now being planned. Of particular interest is the way in which advice is being obtained to fit the hospital to the hospital needs of the borough. The central committee of seven has had placed at its disposal an advisory staff of 60 experts — doctors, nurses, etc. Foreign experts have also been consulted. So wedded is the community to the cooperative idea that not one firm of architects but several are collaborating, including a leading American firm which is acting in a consulting capacity. As this new hospital is to perform advanced social and technical work on a large scale elaborate investigation bureaux and testing departments have been set up. A physician and nurses and others from various departments have been engaged to give expert guidance to architects and engineers in department planning. Five research engineers have been engaged by the city to study the latest technical developments; these engineers are experts in structural engineering, in mechanics, in electrical work, in refrigeration and in transportation. The chief of the department for testing various materials and silencing methods is the head of the Royal Institute of Technology.

Many research studies have been and are being made, for many choices in the past of material and methods have been empirical rather than based upon scientific study. Acoustical studies are being extensively made. Time studies in various hospital activities are being carried out, as in different industries, to judge the cost of upkeep and to appraise efficiency. A laundry expert is making researches on laundry problems and on the decision of whether or not to centralize this work. Actually the centralization or decentralization of all of the various hospital and medical activities is being carefully considered. The kitchen expert has studied various restaurant systems in other countries. It is hoped to raise the standard of medical care in the hospital but not the cost, by standardizing working methods, rooms, equipment, etc. Models are being designed now to become standard throughout Sweden. This program, now well under way, is being approached in a novel and efficient, albeit costly, manner, and the hospital and medical field will be keen to see the final product. Undoubtedly considerable contribution to our knowledge of hospital construction and procedure should result.

Night Operations

Night operations are dangerous. It is strongly suspected if a careful examination of morbidity and mortality statistics covering the so-called emergency operations performed from dark to dawn could be made that these percentages would be startlingly high. Inherent in night surgery are several factors peculiar to this time and type alone. There is the emergency nature of the ailment, often present, just as frequently absent. The fatigue of the operator and his assisting staff, the real or imagined lack of time for a thorough study of the patient, and the spirit of hurry, often

unnecessary, all tend to breaks in technique and the exercise of faulty judgment. Moreover, night is the only time when exacting surgical chiefs permit assistants to perform any solo work. Herein lies at least one explanation for the frequency of night surgery. This factor represents an added danger to the patient. Errors of diagnosis, disaster from infection and hæmorrhage, or any other unfavourable occurrence which befalls the night surgical patient just as much incriminate the chief surgeon as if he himself performed the operation. A glance at the pathologist's report on specimens removed from such patients will reveal to the hospital executive the truth as to the presence or absence of an emergency which required immediate surgical intervention.

The less frequent the night laparotomy, the better will it be for the patient and the hospital.

Medical Societies

The Montreal Medico-Chirurgical Society

The seventh regular monthly meeting of the Montreal Medico-Chirurgical Society for this session, on January 22nd, was the occasion of a pleasant and impressive ceremony. It has always been the custom in the Canadian Medical Association to admit to the class of senior membership those who have been connected with the Association for more than a certain number of years. Also, it has lately been arranged that its past-presidents are to be presented with a special badge. On the occasion referred to, the fulfilment of these customs happened to include five members of this Society of unusual distinction and their presentations were made with fitting formality. The meeting was opened by a paper by Dr. George S. Young, of Toronto, on "The definition of migraine", in which he showed that the exacting administrative duties of his high position in the Association had not impaired his powers as a shrewd clinician, and one with wide experience. His paper called forth interesting comments.

Dr. Young was then translated from the rôle of contributor to that of chairman of the subsequent proceedings. The first item was the conferring of the Canadian Medical Association past-president's badge on Dr. J. C. Meakins, who was introduced by Dr. E. W. Archibald in his characteristically happy manner. Dr. Meakins, in acknowledging the honour said that he felt slightly embarrassed by the fact that he should have been the first recipient of this badge since he had taken part in its design himself.

Medals and certificates of senior membership in the Canadian Medical Association were then conferred on the following: Dr. F. G. Finley, Dr. H. S. Birkett, Dr. H. A. Lafleur, and Dr. W. W. Chipman. The ceremony followed was similar to that in the conferring of special degrees, and a formal introduction by specially chosen speakers was made in each case. The formality of the occasion was greatly enlivened by the personal touches which were added in these introductions.

Dr. Routley then gracefully alluded to its being peculiarly fitting that a society so long and so closely associated with the formation and support of the Association should be the scene of these ceremonies. He dwelt on the various stages of the Association's growth and looked forward to the early completion of the present negotiations regarding federation, which would weld the Association in a body of significant weight in the public health affairs of our Dominion.

Dr. T. H. Leggett, of Ottawa, the President-Elect of the Canadian Medical Association, in a few words assured the Society that the local profession in Ottawa were looking forward to a large attendance at the forthcoming convention in June.

University Notes

University of Manitoba

It was with great regret that the news was received of the resignation of Professor Wm. Boyd, who has accepted a position as Professor of Pathology in the University of Toronto. Dr. Boyd came to Winnipeg in 1915 as Professor of Pathology, and in 1918 became Pathologist to the Winnipeg General Hospital. In the fifteen years of his service he has established a brilliant record as a teacher and author, and his charm of manner, gaiety and capacity for hard work have endeared him alike to student and colleague. His pathological museum is unique as a means of instruction for students and physicians. Needless to say, he will be greatly missed in University circles and from the Winnipeg General Hospital.

The Faculty of Medicine, University of Manitoba, held a three-day course in Obstetrics and Gynæcology for graduates on February 17th, 18th and 19th. The members of the Faculty who led the discussions were Prof. D. S. MacKay, Drs. J. D. McQueen, F. G. MacGuinness, Gordon Chown, L. G. Bell, C. R. Rice, A. Blondal, A. S. Kobrinsky, H. D. Morse, Lennox Arthur, W. F. Abbott, Geo. Brock and Ross Mitchell. In addition to this program the registered members attended as guests meetings of the post-graduate group on Endocrinology, the regular clinical luncheon of the Winnipeg

General Hospital, and the regular meeting of the Winnipeg Medical Society, when there was a discussion on scarlet fever, led by Drs. Dugald McIntyre, E. J. Washington and Ellen F. Taylor.

ROSS MITCHELL

Letters, Notes and Queries

A Correction *re* a Cancer Campaign in British Columbia

To the Editor:

On page 212 of the February issue of the *Journal* there is some information supposedly sent to the *Journal* by Dr. D. E. H. Cleveland. This information is so palpably incorrect that I 'phoned Dr. Cleveland yesterday and he states that this is a transcript from one of our local papers. I would like very much to correct these facts.

In the first place, there is no plan at the present time for a drive being made by the Cancer Foundation in February. Further, I cannot find any authentic information regarding $50,000 having been willed to the funds of the Foundation. It is true that one of our citizens has discussed the question of leaving this sum of money in his will for this purpose.

The bequest by James Inglis Reid referred to has nothing whatever to do with the Cancer Foundation. Mr. Reid has set aside the sum of $25,000 in trust in memory of his son, the late Knox Reid, and under the terms of this bequest the trust company pays to the hospital $1.71 per day towards the hospitalization of two young boys whose parents are endeavouring to meet the hospital bills.

The Vancouver General Hospital is not providing any structure to house the radium at present in safekeeping at one of our local banks. It is true that negotiations have been conducted between the Cancer Foundation and the hospital, but the hospital's decision has been that they cannot finance this building. Further, the statement that there is only $4,000 worth of radium in British Columbia is not correct—there is $105,000 worth. It is true that many of us who are interested in this cancer problem hope that this can be processed for treatment in the near future, but nothing definite in this regard has been arrived at.

Yours very truly,

A. K. HAYWOOD, M.D.,
General Superintendent.

Vancouver General Hospital,
February 11, 1937.

[The item referred to by Dr. Haywood appeared under "British Columbia News" in the *Journal* and was published in good faith. We regret that we admitted matter that was so far from the truth. ED.]

Topics of Current Interest

New Methods of Scientific Publication and Bibliography

The dissemination of new scientific knowledge is second in importance only to research itself. The results of research cannot be of full value in the advancement of science unless they are made known. Publication is the first step in the dissemination of knowledge; the next step is the incorporation of published matter into the bibliography of the subject with which it deals. At present scientific journals cannot always publish all the worthy material offered, and in many cases more prompt publication than now possible may be desirable; also in certain branches of science the bibliographic services are inadequate and may become more so. Fortunately the last statement does not apply to medicine, because the bibliography of the older literature is well covered by the *Index Catalogue* and that of the current literature by the *Quarterly Cumulative Index Medicus*, published by the American Medical Association. A comprehensive and ambitious plan to improve and expand the facilities for scientific publication and bibliography has been inaugurated by Science Service. The new enterprise will centre in the Documentation Division of Science Service, for the work of which the Chemical Foundation has given a grant of $15,000.* Documentation has been defined in the broad sense as "the assembling, classification and distribution of documents of all sorts in all fields of human activity". By way of enlarging the facilities for scientific publication the Documentation Division has developed photographic mechanisms for the prompt issuance of papers and monographs that existing periodicals in various branches of science cannot publish promptly if at all. The plans are based on cooperation with existing periodicals, societies and institutions. Microfilms are made of manuscripts, from which prints can be produced for reading in special machines as well as projection prints for direct reading and general distribution. The films will be permanent and, as orders can be filled as received, no storage of printed stock will be necessary. The process just outlined is used also for copying printed and other matter at small expense. The process is already in use in certain libraries for research purposes and in place of inter-library loans. Thus the Bibliofilm Service of the United States Department of Agriculture is said to give excellent

* Davis, Watson: Microphotographic duplication in the service of science, *Science*, May 1, 1936, **83**: 402; Activities of science service in scientific documentation, *J. Soc. Motion Picture Engineers*, July, 1936, **20**: 77. Draeger, R. H.: Some technical aspects of microphotography, *ibid.*, July, 1936, **20**: 84.

results. Eventually it may prove practicable and economical to publish certain periodicals by microphotographic methods. It is possible to reproduce by microphotography say 150 or more pages of ordinary typescript on an area 3 by 5 inches in extent; from such negatives, prints can be made for reading machines as well as for direct reading. Special attention is given to the development of these methods in cooperation with Dr. R. H. Draeger of the medical corps of the navy. The outlook promises well for the development of new and helpful methods of reproducing reading matter on a large scale. It will be of interest to learn how the problem will be solved of reproducing illustrations, which are such an important feature in medical and other biological literature.

Science Service is interested also in scientific bibliography. It dreams of a complete, centralized international bibliography, of a master file of cards punched and marked to actuate an assorting mechanism, to which is linked a duplicating machine from which bibliographic lists can be delivered instantly to order as desired. Assuming the scheme to be practical, what shall be done with past and present bibliographies? No attempt will be made to discuss details at this time, but the following statement of proposed bibliographic investigations may be of interest: the extent and adequacy of the bibliographies of the scientific literatures of the past; current bibliographic services and the possibility of their cooperation in new plans; the methods of classification; bibliographic mechanisms (including devices for filing and finding, microphotographic cameras, printers); the problems of language and copyrights; methods of international cooperation. Here is a huge task and "the possible inauguration of the bibliographic project is a matter of years and considerable expenditure of money."—*J. Am. M. Ass.*, 1936, **107**: 357.

Abstracts from Current Literature

Medicine

The Metabolic Factor in Chronic Rheumatism with Special Reference to Fibrositis. Gordon, R. G., *Brit. M. J.*, 1936, **2**: 1243.

The author believes that metabolic disturbance is of great etiological importance in all types of chronic rheumatism. This disturbance finds its chief clinical expression in the occurrence of fibrositis, alone, or associated with chronic arthritis. Acute fibrositis is common in young persons, while a usually more chronic form is met with in early old age. The first variety is commonly considered infective in origin, occurring in the same age-group as and sometimes associated with rheumatoid arthritis. Even in the presence of obvious septic foci, and when improvement follows their removal and the institution of vaccine therapy, the author would not have us overlook the possibility of associated thyroid deficiency, or of "imbalance in the autonomic system" which may be corrected by appropriate measures. No disagreement can be found with the statement that chronic arthritis, so often associated with osteoarthritis and responsible for so many of the clinical symptoms, is caused by much the same factors as the latter—strain, trauma, poor vascularity, inefficient circulation and elimination of waste products. The palpable fibrous thickenings found in areas of fibrositis may appear before any clinical symptoms show themselves, and represent a fibrous barrier of resistance laid down round irritative metabolites.

In treatment, search for infective foci and vaccine therapy should not be over-emphasized to the exclusion of measures designed to relieve perhaps more important metabolic defects. Any dietary imbalance should of course be corrected. Thyroid, being frequently deficient, is generally a valuable drug in small doses. The author has found atropine, in vagotonics, ephedrine, in low pressure cases and hyoscine, of value in "autonomic imbalance" cases. The painful fibrous thickenings are best removed by a well-planned course of hydrotherapy or short wave diathermy and firm massage calculated to break down the fibrous nodules. With such treatment much or all of the painful disability may be relieved, sometimes for years.

W. FORD CONNELL

Bromide Intoxication. Barbour, R. F., Pilkington, F. and Sargant, W., *Brit. M. J.*, 1936, **2**: 957.

Bromide is one of the most commonly used drugs, and is frequently used in fairly large doses over very long periods, sometimes without unpleasant effect, but often with the result that dangerous toxic symptoms develop. It is commonly considered that while cumulative toxic effects may occur the warning sign of a bromide rash will first develop; the authors have found skin lesions to be definitely rare, and stress the fact that any toxic symptoms developing in a patient receiving bromide may indicate bromism, the diagnosis being confirmed by the finding of a high blood bromide, together with disappearance of the symptoms on withdrawal of the drug and giving sodium chloride.

It is pointed out that bromide when introduced into the body replaces chloride, the total

blood halide remaining constant. Experimentally, it has been found that 40 per cent replacement in the blood of chlorides proved fatal in animals, and these results are substantially confirmed by clinical observation. Even although death wholly due to bromide over-dosage is rare, patients suffering from milder degrees of intoxication are often in such a weakened condition that they succumb to bronchitis or pneumonia with undue ease. Physical symptoms of intoxication are described —dry skin, furred tongue, tremulous hands, unsteady speech, slurred gait. In some cases, mental symptoms of widely diverse character predominate—hallucinations, severe depression, insomnia, etc., all superimposed perhaps on previous mental symptoms.

It would seem that certain types of patients will show intoxication much more readily than others—particularly old persons, with arteriosclerosis, anæmia, impaired renal efficiency, or organic heart disease. The normal bromide level is 3 mg. per cent, but it can usually be raised to 100 mg. and sometimes 200 without serious symptoms. At over 300 mg. symptoms tend to be severe, and a fatal case is described with bromide of 500, equivalent to a 47 per cent chloride replacement.

It is desirable that there should be more widespread recognition of the common occurrence of dangerous cumulative effects from this drug. The treatment of intoxication, when it is suspected, is simple. The drug being stopped, large amounts of fluid are given by mouth, together with about 20 to 40 grains of sodium chloride every four hours.

W. FORD CONNELL

Surgery

Perforated Peptic Ulcer. Bailey, H., *The Lancet*, 1936, **2**: 249.

This is an analysis of 107 cases of perforated peptic ulcers. Of these, 10 were in women.

The pulse rate is comparatively slow in early cases. Sixty-three cases were under eight hours, and 51 patients had a pulse rate of about 80. The author has examined patients five minutes, fifteen minutes, and thirty minutes after perforation, and found that the pulse rates were 62, 96 and 80 respectively.

The sooner the operation is performed the better. Forty per cent of his patients were perforated for twelve hours before admission, 15 per cent over twenty hours, and of these, 8 died. "A less appreciated fact is that most of the patients who died from shock were early cases."

Of the 107 cases, 12 were over sixty, 2 were eighty or over and of this 12 only 3 survived. There were 29 deaths, 8 from chest complications, 6 cases of general peritonitis, 5 of shock, 3 of hæmatemesis, 2 of pulmonary embolus, 2 of subphrenic abscess, 2 of intestinal obstruction, and 1 of uræmia (cystic kidneys).

Hæmatemesis as a complication of perforation convalescence is very serious. The three reported cases were all in women with hour-glass stomach. Bailey recommends gastro-gastrostomy in addition to closure of the perforation.

Treatment. — Simple suture reinforced by omental graft is the method of choice. Chest complications and primary shock are the two most fatal complications. The patient is prepared by morphine, gr. ¼, and continuous intravenous saline during operation. The abdomen is opened under local anæsthesia in the mid-line. On reaching the peritoneum evipan is injected into the rubber tube of the intravenous apparatus. This gives plenty of time for suturing the perforation, placing a drainage tube in the recto-mesical pouch, and bringing it out through a supra-pubic stab wound. Bailey favours drainage, and gives examples of fatality that might have been avoided by drainage.

S. A. MCFETRIDGE

Partial Gastrectomy in acute Perforated Peptic Ulcer. Judin, S. S., *Surg., Gyn. & Obst.*, 1937, **64**: 63.

The author reports his observations and treatment of 426 new cases, all of which were treated as emergencies. Ninety per cent of the patients were males and 90 per cent of this series occurred in the duodenum. Perforations took place more frequently in the winter season, and the incidence was highest when avitaminosis was most prevalent. There was a noticeable increase of occurrence in the hours between 12 noon and 5 p.m. and between 11 p.m. and midnight; 3 p.m. was the hour of greatest danger. The diagnosis was based upon clinical examination with x-ray confirmation by the presence of gas levels in the upper abdomen; in 95 per cent of cases they were able to obtain a positive history of preceding digestive disturbances which might be interpreted as peptic ulcer. Judin uses ether induction, with local anæsthesia, by preference, but resorts to 1 per cent solution of percain spinal anæsthesia if a chief anæsthetist is not available.

Resections were performed in 80 per cent of these cases, the vast majority of which were treated by the Billroth 1 method, the remainder by the Polya-Balfour method. All younger persons were resected if the stomach or duodenum were freely movable. The mortality was 7.8 per cent. In the remaining 20 per cent suture of the ulcer with gastroenterostomy was performed, because of the age of the patient or ulcer or the presence of advanced peritonitis, with a mortality of 32.2 per cent.

FRANK DORRANCE

The Transfer of Tumour Cells by the Surgical Knife. Saphir, O., *Surg., Gyn. & Obst.*, 1936, **63**: 775.

Many surgeons discard the scalpel and all other instruments after biopsy and after the removal of malignant tissue, and also change their gloves before proceeding to handle apparent healthy tissue. In all cases where the instruments were examined many viable tumour cells were found adherent to the knife-blade, comparable to the presence of erythrocytes on a blood smear of average thickness. These results were particularly noted in operations upon malignant tumours in the breast.

FRANK DORRANCE

Obstetrics and Gynæcology

The Cell Volume following Delivery and its Relation to Blood Loss and Post-partum Infection. Pastore, J. B., *Am. J. Obst. & Gyn.*, 1936, **32**: 859.

The incidence of puerperal infection is proportional to the loss of blood. This increases with the weight of the patient, and the seriousness of the hæmorrhage is inversely proportional to the body weight. For blood losses less than 0.3 per cent of body weight, the incidence of infection remains fairly constant around 6.5 per cent. Above this value it rises rapidly until the figure reaches 0.7 per cent. This rise increases the incidence of infection by over 100 per cent. For losses between 0.7 per cent and 1.0 per cent the incidence of infection is again constant. A secondary rise begins at the 1.0 per cent loss of blood, and increases the incidence to 31.8 per cent for an average loss of blood of 1.5 per cent.

When the loss is less than 0.3 per cent there is an increase in cell volume by the third post-partum day, due to dehydration of the blood following delivery. Such a rise in cell volume seems to be necessary for an ideal puerperium. With larger losses of blood, in spite of the fact that the puerperium is considered afebrile, fluctuations in temperature between 37° and 38° C. are usually noted. When the loss is between 0.7 and 1.1 per cent the bone marrow may be stimulated, the maximum stimulation being obtained at 1.1 per cent.

Patients with a cell volume of 40 per cent or over on the third post-partum day had an incidence of infection of 4.5 per cent; with a cell volume between 30 and 40 per cent the incidence was 7.5 per cent; below 30 per cent cell volume the incidence increased to 31 per cent. With a calculated cell volume of below 30 per cent blood transfusion should be given within the first twelve hours following delivery. The amount of blood given depends on the weight of the patient, the loss of blood and the cell volume before delivery. ROSS MITCHELL

Primary Malignant Diseases of the Vulva, with special Reference to Treatment by Operation. Blair-Bell, the late W. and Datnow, M. M., *J. Obst. & Gyn. of Brit. Emp.*, 1936, **43**: 755.

Malignant disease of the vulva occurs as squamous-cell carcinoma in over 90 per cent of cases. Adenocarcinoma arising in the Bartholinian region occurs in about 5 per cent; therefore the authors discuss in detail the squamous cell type. About 2 to 3 per cent of all genital carcinomas are on the vulva, and are found most commonly between the ages of 50 and 70 years. Parity does not appear to have any relation to the disease. The labia majora and clitoris claim the greatest number. Leukoplakia is a predisposing cause. The disease may be papillary (cauliflower-like), ulcerative, or deeply invasive with little superficial evidence of the disease. Pruritus and burning are early clinical symptoms; later are lumps or swelling where there is a superficial ulceration. Syphilis and tuberculosis must be excluded. The ulcerative and invasive types rapidly involve the inguinal and femoral glands; 50 per cent show such involvement in the second six months of the disease. Untreated patients die within two years, and only 15 per cent of those operated on survive for five years. The absolute cure rate is 7 per cent.

Kehrer points out that the superficial inguinal lymphatic glands on the two sides are anastomotic and malignant disease on one side infects the inguinal glands on the other. The superficial lymphatic glands on either side are also connected with the deep inguinal, the obturator, and the pelvic (iliac) glands of the same side. The radical operation removes the superficial inguinal and femoral glands. The greatest trouble is with infection after operation. Some operators prefer two stages, removing the inguinal and femoral glands first.

P. J. KEARNS

Traumatic Gynatresia. Kassebohm, F. A. and Schrieber, M. J., *Am. J. Obst. & Gyn.*, 1936, **32**: 869.

Five cases of irreparable vaginal atresia are reported due to packing a lacerated abraded vagina with iodoform gauze. Two cases of almost complete failure of cervical effacement and absolute default of dilatation of the external os in multiparæ are also reported, due to electrocoagulation of the endocervical mucosa for chronic endocervicitis. ROSS MITCHELL

Pædiatrics

Mandelic Acid in the Treatment of Pyelitis in Childhood. Newns, G. H. and Wilson, R., *The Lancet*, 1936, **2**: 1087.

These authors report the treatment of 36 cases of pyelitis with mandelic acid; all in

children under twelve years of age. In most of the cases mandelic acid together with sodium bicarbonate was used, and was given in doses of 15 to 30 grains a day according to the age of the child. As a rule half this dose of ammonium chloride was given to acidify the urine. In 6 cases "Neoket" (a mixture of mandelic acid and acid sodium phosphate) was used, and in 4, "Mandelix", a solution of ammonium mandelate.

The authors conclude that mandelic acid appears to be an effective remedy for *B. coli* pyelitis in children, the most striking feature being the rapidity with which the urine is rendered sterile. They treated both infants and older children without harm. They found also that pyrexia and albuminuria were not contraindications to its use. Relapses took place in 9 cases out of the 36 cases treated. Three of these were in patients with abnormalities of the urinary tract, while 5 had infections with organisms other than *B. coli* which reacted less well to mandelic acid. Some of these relapses might have been prevented if the exhibition of mandelic acid had been more prolonged. Treatment should be continued for at least a week after the urine has become sterile, and probably longer, especially in chronic cases. Further study will be necessary before it will be possible to say whether the cures with mandelic acid are permanent or not.

JOHN NICHOLLS

Ophthalmology

Contribution to the Etiology of Familial Affections of the Optic Nerve. Rifat, A., *Ann. d'Ocul.*, 1936, **173**: 702.

Subacute retro-bulbar neuritis of Leber and familial and total atrophy of the optic nerve have an obscure etiology, and up to now it has not been possible to attribute any precise cause to these diseases. In the genesis of familial disease the rôle of syphilis has recently been stressed by Leredde, who because of its chronic character gives it a preponderant influence in the etiology of familial disease. We also know that syphilis is the only hereditary and congenital infection that is transmitted to several generations. Some observations exist in the literature where Leber's disease might be considered as a manifestation of hereditary syphilis. Buisson, in his thesis of 1899, reported a brother and sister presenting a classical picture of this condition, where 5 other brothers and sisters had died young, and 2 others were born dead. Here syphilis can only be suspected, but it looks most probable. In 1873 Schmidt Rimpler reported a subacute retro-bulbar neuritis where the patient suffered from pigmentary retinitis.

The author has examined a family recently where there were 6 children living; three, beside a typical pigmentary retinitis, presented unquestionable signs of hereditary syphilis. Seven observations in detail are then given. Rifat believes that familial affections of the optic nerve appear in three forms.— (*a*) Subacute retro-bulbar neuritis of Leber, in which the lesions affect only the macular branch of the optic nerve. The onset is usually around the age of 20 to 30 years, and the affection progresses for a year or two, then rests stationary, and is never cured. (*b*) Familial atrophy of the optic nerve, which affects all the branches of the optic nerve. Absence of a central scotoma and peripheral shrinking of the visual field differentiates this from the disease of Leber. There is partial blindness which rarely progresses to completeness, and this is associated with mental retardation. (*c*) Familial congenital atrophy of the optic nerve—a condition which exists at birth and usually involves several members of the same family in the same and in successive generations. Syphilis should be searched for in a systematic manner by all the methods at our disposal in all cases of familial disease of vision.

S. HANFORD MCKEE

Orthoptic Treatment of Strabismus. Bressler, J. L., *Am. J. Ophth.*, 1936, **19**: 989.

The value of orthoptic treatment for the correction of strabismus has begun to assume such importance that a knowledge of its methods and routine is becoming increasingly necessary as a part of the ophthalmologist's equipment. Orthoptic treatment is frequently useful in correcting strabismus as the only means employed. Also it may be combined with surgery, when it commonly leads to better results than surgery alone. A word of warning and advice will not be out of place, as it would not be well to expect every case of strabismus selected for orthoptic treatment to respond rapidly and end in a cure, for disappointment with the results may follow. Cases must be carefully selected for this method of treatment, and the difficulties of each case must be carefully studied.

The data presented in this paper tend to show that orthoptic treatment should not be judged by percentages of cures of strabismus with non-operative treatment alone, but should also be considered for its value both before and after surgical correction for deviation.

S. HANFORD MCKEE

The Differential Diagnosis of Orbital Gumma. Wolfsohn-Jaffe, E., *Brit. J. Ophth.*, 1936, **20**: 626.

The differential diagnosis of retro-bulbar tumours of the orbit is often very difficult.

Frequently only an exophthalmos and secondary changes in the eye-ball suggest the presence, deep in the orbit, of a pathological process which cannot be detected by inspection or by palpation or by x-rays. In such cases any auxiliary means of diagnosis which may help to clarify the question as to whether there is a benign or malignant tumour or a chronic inflammatory process may certainly be welcomed.

The details of a case are described in which Gutmann's piezometer, an apparatus for measuring the displaceability of the eyeball into the orbit, was used. A case of orbital gumma was examined with the piezometer during the course of the affection, and it was found that the displaceability of the globe into the orbit diminished during the healing of the gumma and remained later permanently less than normal. Diminishing piezometer values during the course of an anti-syphilis cure indicate resorption and cicatrization of the pathological retro-bulbar process, as expressed in the form of a fibrous induration, and contribute to the diagnosis of syphilis.

S. HANFORD MCKEE

Urology

The Relation of the Parathyroid Glands to Urinary Lithiasis. Barney, J. D. and Mintz, E. R., *J. Urol.*, 1936, **36**: 159.

As a result of investigations by many men and from their own experience the authors have come to agree with Albright that "Hyperparathyroidism is a sufficiently frequent cause of renal stone formation that its presence must be ruled in or out in every case of this disease", and so they suggest that a study of the blood for its calcium and phosphorus content should be done almost routinely in cases of urinary lithiasis. A blood calcium of 11.5 mg. per cent or above suggests hyperparathyroidism. In a series of 288 cases of urinary lithiasis in which blood calcium and phosphorus were studied, 12 cases, or 4.16 per cent, of hyperparathyroidism proved by operation were found. In a series of 29 cases of hyperparathyroidism proved at operation 20 cases, or 68.9 per cent, were associated with urinary calculi. As a result of a study of 35 cases of bilateral stones together with the blood chemistry, the authors have come to believe that parathyroidism does not seem to be a factor in the production of bilateral lithiasis.

In the matter of treatment, the authors state that it is largely a matter of surgical judgment as to whether the parathyroid tumour or the urinary stone be operated on first. They believe that in the presence of free kidney drainage and but slight renal damage it is the better policy to first remove the underlying cause of the metabolic dysfunction. However, if one or both kidneys are badly damaged or blocked by stone it is the better procedure to improve the urological status so far as possible. Employing the above as basic principles, the operation for the removal of a parathyroid tumour (29 cases) has as yet resulted in no mortality at the Massachusetts General Hospital, though it may be and often is an operation of the first magnitude, and for this reason it is wiser to have the urinary tract put into the best possible condition. Following operation the blood calcium drops, often quite rapidly, and at times so low that tetany occurs. Eventually, however, it reaches a point within normal limits, and as far as the authors have observed it remains there. At the same time the authors have noted no cases of recurrent lithiasis after the parathyroid tumour and the urinary stone have both been successfully removed.

N. E. BERRY

The Relationship between the Chemical Composition of Renal Calculi and Associated Bacteria. Priestly, J. T. and Osterberg, A. E., *J. Urol.*, 1936, **36**: 447.

This study was undertaken with the idea of correlating some of the clinical data presented by patients who have urinary calculi. The authors point out that stones of different chemical composition are not found with equal relative frequency in all parts of the urinary tract. The greater percentage of oxalate stones were removed from the kidney, (82.8 per cent). This was less true of phosphatic calculi (61.9 per cent), and, in contrast, slightly more urate stones were removed from the bladder (51.7 per cent) than were removed from the upper part of the urinary tract. The clinical history in the latter group would tend to contradict this, if one were considering the formation of such calculi.

Although 85 per cent of calculi are unilateral, yet in the cases which have bilateral occurrence the urate and phosphate stones predominate. Many stones seem definitely related to certain types of infection, whereas other calculi commonly form in the presence of sterile urine. Phosphate stones are not only more commonly found in an infected urine but bacteria are more frequently demonstrated in this type of calculus than in stones composed of oxalates. The association of *Proteus ammoniæ* with phosphatic calculi is striking, while *Escherichia coli* has a relatively high incidence with all three forms of stones. In the tabulations the wide variety of organisms that may be present indicates that under the proper circumstances many different types of bacteria may be associated with the precipitation of stones. The studies of the pH of the urine reveal that a normal range is between 5.0 and 7.5, with an average of approximately 6.0, and that certain bacterial infections

may fix the pH to a large extent. Tabulations are included on this aspect of calculous disease.

J. V. BERRY

Neurology and Psychiatry

Occlusion of the Aqueduct of Sylvius by Neoplastic and Non-neoplastic Processes with a Rational Surgical Treatment for Relief of the Resultant Obstructive Hydrocephalus. Stookey, B. and Scarff, J., *Bull. Neurol. Inst. N.Y.*, 1936, **5**: 348.

A review of 16 cases of non-neoplastic aqueduct stenosis and 6 cases of periaqueduct tumour reported in the literature failed to show a single instance of successful removal of the obstructive process or relief of the obstructed ventricular system. The typical clinical picture of chronic aqueduct insufficiency is that of a twelve year old child of either sex, with a history of symptoms for eight or nine years, who complains of headaches increasing in severity, accompanied by vomiting, and associated with unsteadiness in gait and, occasionally, convulsive seizures. A typical case of periaqueduct tumour is presented by a boy of about fourteen years, with a dyspituitary habitus, some hypersomnolence; cerebellar signs, usually bilateral, with little or no nystagmus; visual disturbances; increased reflexes, and bilateral Babinski. Ventriculograms are of great value in the diagnosis of aqueduct obstruction, yet even with this additional help a diagnosis of tumour in the posterior fossa is frequently made when the lesion is really at the aqueduct. The authors describe a new surgical procedure, which consists of draining the obstructed cerebrospinal fluid out of the ventricles directly into the large subarachnoid cisternæ at the base of the brain by means of openings through the lamina terminalis and the floor of the third ventricle. Six cases have been treated by this method. Three of these patients who belonged to the non-neoplastic group appear to be permanently cured. The fourth surviving case, which belongs to the neoplastic group, has been improved.

FRANK TURNBULL

Glioblastoma. A Point of View concerning Treatment. McKenzie, K. G., *Arch. Neurol. & Psychiat.*, 1936, **36**: 542.

It is held by certain authorities that operation for the treatment of glioblastoma is futile and better left undone if one can be reasonably certain of the diagnosis before operation. Such a nihilistic point of view is usually formed after a few experiences with the treatment of these cases by simple external decompression. Commonly such treatment is followed by a prolonged period when the patient, deformed by a bulging decompression and flap, has total disability and is a financial burden on his relatives or on the community. McKenzie holds that operation in most cases of glioblastoma is worth while provided that the tumour is resected in a sufficiently radical manner as to leave a large cavity. This usually involves the removal of a mass of tumour and overlying brain, approximately the size of an orange. When this is done all tension is relieved, and no attempt is made to eradicate the deeper parts of the tumour. The dura is then carefully sutured. When a satisfactory internal decompression is performed in this manner the danger of immediate death from operation is not greater than that from simple external decompression. The space left after this resection eventually fills up again with tumour and symptoms return, but life is not then unduly prolonged because the dura has been closed tightly. Such an operation provides the patient with a period of several months during which he may carry on in a satisfactory manner with little or no disability and without deformity.

FRANK TURNBULL

Dermatology

Les épithéliomas cutanés post-traumatiques. Marques, J. F., *Annales de dermatol. et de syphil.*, 1936, **7**: 1004.

Marques reports 2 cases of his own, and compiles a series of 26 cases from recent literature, of carcinoma of the skin following directly upon a single local injury. He excludes cases developing after repeated trauma. Such single local injuries may be produced in a variety of ways. Among those quoted are pricks with wire or splinters of wood, scratches, razor-nicks, burns from hot liquids, various chemicals, hot metals, cinders, cigar-butts, blows, etc.

It was noticeable that the majority of the cases occurred in patients under 45 years of age. Only 3 cases occurred in women. Eight cases only followed burns, and of these only 4 were burns from substances such as tar, commonly considered as predisposing to cancer. The remainder were due to mechanical trauma, especially in which a foreign body penetrated the skin. Solution of continuity of the integument was apparently a necessary feature of the injury. In 22 cases the lesion remained open from the time of injury until the diagnosis of malignancy was made. In the great majority of the cases the evolution of cancer was so rapid as to justify the term "acute cancer". In most cases the elapsed time between the trauma and the histological diagnosis was less than six months.

Four cancers were basal-cell, 2 were mixed-cell, and the remainder of the prickle-cell variety. No reference was made as to the grade of malignancy. A striking feature noted was that in areas where basal-cell lesions

ordinarily predominate the lesions following single trauma were prickle-cell cancers.

The author also refers to a further series of 23 cases reported by various authors which he has been unable to tabulate with the cases already referred to. In these original and complete data were not available, but such as were furnished appeared to justify placing them under the same heading.

The author concludes by presenting his views on the manner of production of cancer following directly upon single unrepeated trauma, harmonizing them with the views already held upon the etiology of cancer resulting from prolonged exposure to cancerogenic substances, chronic irritation, infection, and single frequently repeated trauma.

D. E. H. CLEVELAND

Xeroderma Pigmentosum: An Inherited Disease due to Recessive Determiners. Macklin, M. T., *Arch. Derm. & Syph.*, 1936, **34**: 656.

This study of cases of xeroderma pigmentosum collected from the literature shows that many statements made in texts of dermatology concerning this disease are erroneous. It shows that the disease is inherited, and is dependent upon recessive determiners, so that children affected with the condition are born of normal parents. It also shows that the disease does not tend to affect one sex only in the family and leave the other sex unscathed, a misconception which has been fostered by the repetition in dermatology texts. Nor, having affected one child in a family, does it tend to affect all subsequent children of that sex in the family. Numerous families are found in which both sexes are affected, and in which normal children of both sexes occur; this again is contrary to the statements in many textbooks. Moreover, although it is inherited, because of the mode of its transmission, (recessive) and because of the small size of human families, the majority of families (60 per cent) have but one child affected. Only 40 per cent of families have two or more children affected with the condition. In spite of this, it is unwise to say that any family will have only one affected. Consanguineous marriages were found in over one-fifth of the parents of children affected with xeroderma pigmentosum.

MADGE THURLOW MACKLIN

Physiology and Biochemistry

The Metabolism of Human Erythroblasts. Kempner, W., *J. Clin. Investigation*, 1936, **15**: 679.

The metabolism of human nucleated red cells has not previously been determined because of the difficulty of procuring material. The author recently came across a patient with erythroblastic anæmia who had an extremely high proportion of nucleated red blood cells (130,000 — 364,000 to 1,800,000 — 3,500,000 of non-nucleated erthrocytes in 1 c.mm. of blood), who thus afforded an excellent opportunity for study. He measured the metabolism of the nucleated cells manometrically. These showed a very high oxidative and fermentative metabolism. Their respiration was approximately 200 times greater than that of normal human erythrocytes, 100 times greater than the respiration of the red blood cells of anæmic patients, and about 20 times greater than that of the nucleated red blood cells of geese. In the case of the erythroblasts, as in human inflammatory exudates, in leucocytes, and in non-nucleated red blood cells, respiration is not sufficient to cause the lactic acid formed to disappear, but the ratio of respiration metabolism to glycolytic metabolism, as compared with that of non-nucleated red blood cells, is shifted decidedly in favour of the respiration metabolism.

The anaerobic lactic acid formation of erythroblasts is about 90 to 100 times greater than that of normal human blood cells and of the normal erythrocytes of geese, and 25 times greater than that of the erythrocytes of geese with marked anæmia. The anaerobic lactic acid formation is of the same order of magnitude as that found in the youngest embryonic tissue.

JOHN NICHOLLS

Therapeutics

The Problem of Rheumatism and Arthritis. A review of American and English literature for 1935. (Third Rheumatism Review). Hench, P. S., Bauer, W., Fletcher, A. A., Ghrist, D., Hall, F. and White, T. P., *Ann. Int. Med.*, 1936, **10**: 754.

Atrophic arthritis.—The patient and not just the disease must be treated. Whatever physiological abnormality, whatever coincident or related infection, endocrine or metabolic disturbance, whatever postural or occupational strain a patient presents should be corrected as far as possible in order to help the patient cure himself of a disease which the physician cannot or has not yet learned how to cure. The removal of foci of infection alone is rarely successful. One should distinguish between unwalled-off infected foci that may produce systemic disease and walled-off infections such as dental root cysts, which may be harmless. Many infected foci may be the result not the cause of the disease. In dealing with infected sinuses conservative treatment should be thoroughly tried before operation. Relief of arthritis following operations may sometimes be due to a non-specific post-operative effect, and is not necessarily due to the removal of the infected focus. The importance of secondary colon infections is often

exaggerated. Streptococcal vaccines are of unproved value, generally harmless, have a psychotherapeutic value, and serve to bring the patient to the physician's office for more important treatment and control. The majority of those treating arthritis favour no particular dietary restrictions, prescribe a generous intake of food, except when a reduction of the trauma due to obesity is required, and believe that most patients do badly on prolonged dieting or starvation. Many believe that vitamin deficiency plays no part in the disease. Some restrict concentrated carbohydrates. This tends to provoke a negative water balance, which reduces swelling and inflammation. Treatment by gold salts, intravenously or intramuscularly, has a mortality rate of approximately 3 per cent. The results do not warrant their use. The results obtained by the use of massive doses of vitamin D were not unlike those obtained by other methods, and a conservative attitude toward such therapy should be adopted. Prolonged rest in bed is necessary for acutely swollen joints, but in chronic cases the problem is one of rest and exercise. The patient may overdo on rest, with resultant atrophy and ankylosis, but the majority overdo on exercise to "keep the joints limber". Whenever possible the arthritic patient should receive physical therapy in three ways: (1) by daily home measures; (2) professional physiotherapy three or more times a week; and (3) annual or semi-annual visits to a spa or other institution for treatment combined with the advantages of a vacation. Roentgen therapy and fever therapy are really of little value in atrophic arthritis and should not be used to the exclusion of other treatments. Sympathectomy is not justified by results. Manipulation is a valuable method used insufficiently. As a rule atrophic arthritis becomes inactive relatively slowly, even under the most successful treatment. Particular significance may therefore be attached to the dramatic sudden remissions that may accompany intercurrent jaundice. In the majority of cases the remission was complete, and lasted weeks or months, occasionally longer. An intensive study of this phenomenon may result in a method of treatment similarly dramatic and prompt.

Hypertrophic arthritis.—Perhaps the most important part of treatment is to explain to the patient the differences in the nature, particularly in the prognosis, of the disease he actually has from that which he thinks he has, and to assure him that his disease in not essentially an ankylosing, severely-crippling, progressive disease. This done, many patients neither ask nor accept other treatment, but bear their difficulty philosophically without the "nuisance of treatment".

H. GODFREY BIRD

Pathology and Experimental Medicine

The Artificial Induction of Subcutaneous Nodules in Patients with Rheumatic Fever. Massell, B. F., Mote, J. R. and Jones, T. D., *J. Clin. Investigation,* 1937, **16**: 125.

Drewit first hypothesized that trauma was involved in the question of the subcutaneous nodules of rheumatic fever, because of their common occurrence over bony prominences. The authors report the result of their attempts to produce such nodules. Their studies were carried out on 116 patients. Sixty patients had rheumatic fever; 22 had active or subsiding chorea; 34 were persons with diseases other than rheumatic fever.

Massell and his associates found that the injection of 10 patients' own blood into their subcutaneous tissues frequently resulted in the formation of subcutaneous nodules in the areas injected which were clinically indistinguishable from nodules that occurred spontaneously. The same procedure carried out in 24 presumably non-rheumatic persons resulted in nodule formation in only a single instance.

JOHN NICHOLLS

The Pathology of Spontaneous and Induced Subcutaneous Nodules in Rheumatic Fever. Mote, J. R., Massell, B. F. and Jones, T. D., *J. Clin. Investigation,* 1937, **16**: 129.

The authors describe in a very detailed manner the pathology of spontaneous and artificially induced subcutaneous nodules in rheumatic fever. The latter were produced by the injection of the patients' own blood. The histological picture in them was very similar to, if not identical with, that of the nodules occurring spontaneously. There are, however, certain alterations in the induced nodules, due probably to the manner of their production, such as damage and distortion of tissue from the injection. These alterations or differences were three. First, the areas of collagen alteration are much larger and the strands of the collagen are considerably wider than is the case with most spontaneous nodules. Secondly, an appreciable amount of blood is introduced intercellularly, which results in a greater phagocytic cell response than is the case in most of the young spontaneous nodules. Thirdly, there is a considerable amount of intercellular and interfibrillar amorphous precipitate in these lesions, apparently the result of introducing large amounts of blood into the tissue spaces. Although these artefactual characteristics are such that they usually permit of differentiation between young induced and young spontaneous nodules, the type of tissue response is in most instances similar and in some cases indistinguishable. No difference of

structure between the two types can be determined in the older age group.

JOHN NICHOLLS

Hygiene and Public Health

Syphilis Mortality Declines Among White Persons, *Bull. Metropolitan Life Ins. Co.,* 1936, **17**: 3.

Comparing the years 1911 to 1915 and 1931 to 1935 the combined death rate from syphilis, locomotor ataxia and general paralysis of the insane has declined about 45 per cent among the white policy holders of the Metropolitan Life Insurance Co.; among the coloured policy holders, on the other hand, the mortality from these diseases has increased 37.7 per cent among males and 13.9 per cent among females.

Syphilis is a major public health problem. Mortality statistics, as is well known, do not reflect the gravity of the problem at all, because syphilis is greatly understated on death certificates. It is estimated that about 7,000,000 people in the United States have syphilis at any one time. The decline in syphilis among white persons may be attributed to the increased use of protective methods, earlier and more adequate treatment, and the increased number of medical examinations which may reveal unsuspected cases. The control of the disease rests on an increase of these measures, particularly an increase in the facilities for treatment.

FRANK G. PEDLEY

Clinical Studies on Lead Absorption in the Human: III. Blood Pressure Observations. Belknap, E. L., *J. Ind. Hyg. & Toxicol.,* 1936, **18**: 380.

The general teaching is that lead poisoning is a cause of hypertension. Belknap finds no evidence of this. In a series of 81 men who showed evidences of heavy lead absorption and who were clinically observed over periods of from 1 to 5 years the tendency of the blood pressures was to fall slightly. These men were selected on the basis of showing lead absorption, and no men showing this were excluded. The median blood pressure readings of these men were all within normal limits for each age group.

FRANK G. PEDLEY

As good almost kill a man as kill a good book. Many a man lives a burden to the earth; but a good book is the precious life blood of a master spirit, embalmed and treasured up on purpose, to a life beyond death.—Milton.

Obituaries

Dr. Francis Alexander Carron Scrimger, V.C., of Montreal, died suddenly at his home on February 13, 1937. He was in his fifty-seventh year.

Dr. Scrimger was born in Montreal in 1878 the son of the late Rev. John Scrimger, D.D., Principal of the Montreal Presbyterian College. He was educated at McGill University and graduated in both Arts and Medicine, obtaining his B.A. in 1901, with first class honours in biology, and his M.D., C.D. in 1905. He served one year in medicine, and two years as house surgeon, etc., at the Royal Victoria Hospital under the late Dr. James Bell, and in 1909 took a post-graduate course in Europe, devoting the greater part of his time to pathology under the direction of Dr. Schmorl at the government laboratories in Dresden. Later he went to Vienna and Berlin and served under Dr. Bier, personal surgeon to the then Kaiser. On his return to Montreal, Dr. Scrimger entered into private practice as a specialist in surgery and was associated with Dr. Edward Archibald in a good deal of research work. He became associate in surgery at the Royal Victoria Hospital and acted as medical officer for the Montreal Heavy Brigade, under the late Lieut.-Col. Lacey R. Johnson.

On the outbreak of war Dr. Scrimger was appointed medical examiner for the Artillery and later went to Valcartier, where he was offered the post of medical officer of the 14th Battalion Royal Montreal Regiment by Lieut.-Col. F. S. Meighen. He promptly accepted and returning to Montreal for two days closed up his civilian affairs and joined the regiment, to go overseas with it. On Salisbury Plain he was attached to the staff of Gen. Jones, head of the Canadian Army Medical Corps, who wanted to keep the clever young surgeon with him. But Dr. Scrimger wanted to get to the front, and although he was delayed a little while by illness he finally succeeded in rejoining his battalion just before the second battle of Ypres, when the first poison gas attack by the Germans was made.

It was during those April days of fierce fighting that Dr. Scrimger won the Victoria Cross, which was to be later presented to him personally by King George V at Windsor Castle on July 22, 1915. Capt. Scrimger, as he then was, had charge of a field dressing station at Wieltje, about two miles north of Ypres. Beyond — at Kitchener's Wood, St. Julien and other places — the Canadian troops were fighting desperately to stem the German advance, and the station was filled with wounded soldiers and victims of gas. Day and night Dr. Scrimger displayed his devotion to duty, and his gallantry reached its peak when the German artillery started to shell the farm buildings in which the headquarters staff of the 3rd Brigade was quartered, and part of which was being used as an advanced dressing station. The shelling became so heavy that the medical officer had the wounded removed to a place of greater safety. The building was set on fire and General Turner ordered his staff to scatter, fearing that a direct hit would wipe them all out. Capt. H. F. McDonald, now Brig.-Gen. McDonald, chairman of the Canadian Pensions Commission, Ottawa, was then a member of the staff, and was at the time in front of the building and was hit in the neck and the shoulder. He was dragged into the building and Capt. Scrimger dressed his wounds, but the shelling got worse and Capt. McDonald asked to be taken out into the open where he would take his chance, for the staff had abandoned the building and his was apparently a hopeless case. What followed is best described in Captain McDonald's own words, as given to *The Montreal Star's* London correspondent on July 15, 1915:

"But Capt. Scrimger carried me out, and down to a moat 50 feet in front, where we lay, half in water. Capt. Scrimger curled himself round my

wounded head and shoulder, to protect me from heavy shell fire, at obvious peril to his own life. He stayed with me till the firing slackened, then the stretcher bearers carried me to the dressing station. This however, is only one of many incidents of Capt. Scrimger's heroism in those awful three days. No man ever better deserved the soldier's highest honour.''

As a matter of fact this was only part of the story, for it subsequently became known that several eight-inch shells fell within a very short distance of them, and Capt. Scrimger held back the sliding mud bank which threatened to bury them, while to add to it all the burning barn contained thousands of rounds of rifle ammunition which exploded in all directions. Miraculously neither officer was hit.

Capt. Scrimger subsequently commanded No. 1 General Hospital in France, and had charge, later, of Granville Hospital, England, and No. 3 General Hospital, Boulogne, France, which was a McGill unit, the doctor having by this time been raised to the rank of Lieut.-Colonel. He returned to Canada on May 7, 1919, and on demobilization rejoined the staff of the Royal Victoria Hospital, and in 1932, was advanced to the rank of surgeon, in January, 1936, becoming director of the department of surgery at the hospital and associate professor of surgery at McGill University.

Dr. Scrimger married Ellen Carpenter, who survives him, as does one son, Alexander, three daughters, the Misses Jean, Charlotte and Elizabeth Scrimger; two sisters, Mrs. Walter Lyman and Mrs. Harold Skelton, of Montreal; and one brother, Rev. J. Tudor Scrimger, of Norham-on-Tweed, Northumberland, Eng.

AN APPRECIATION

The death of Dr. Scrimger, so sudden and so tragic, is a very grave loss to the Royal Victoria Hospital, to McGill University and to the community. He was possessed of unusual qualities and of quite unusual capacity. His scholastic career was distinguished, he was always the student, but in addition he was a very practical man, and this was shown in his great ability as an operating surgeon and also as an organizer in the work of his department in the hospital and in the faculty. He was considered by the students as one of their best teachers. In addition he had marked ability as a research worker and made several original contributions to the science of surgery as well as to its handicraft.

In disposition he was essentially quiet and unaggressive, but was also tenacious of his own opinion, once formed, and in action firm and unhesitating. He possessed a great capacity for friendship and had a wide circle of close friends. He was particularly valued as a consultant by his medical confrères because of his wide knowledge, his diagnostic ability and his general soundness and clearness of view.

On this continent and in the Old Country he enjoyed a high reputation as a scientific surgeon. This was widely recognized by his membership in the most important and also the most exclusive surgical societies. There will be grief far beyond the limits of his own city among a host of friends and admirers in Canada and abroad. Many of us, his confrères, feel his death as a deep personal loss.

EDWARD ARCHIBALD

Dr. Ingersoll Olmsted, of Hamilton, Ont., the well known surgeon, died on November 12, 1936, in his seventy-third year.

Dr. Olmsted was born in Ancaster and attended public school there. He later attended the Collegiate in Hamilton, now Central Collegiate Institute, and graduated from the University of Toronto (M.B.) in 1887. He then went to Philadelphia and became an intern in the German hospital, returning to Hamilton in 1888.

In 1893 he was offered the assistant professorship in bacteriology in the University of Pennsylvania. It was here, using himself as a subject for an experiment, that he developed an infection, and, back in Hamilton, Dr. Malloch removed his right leg below the knee. He resigned his position at the university, and journeyed to Europe to acquaint himself with the latest in surgery. Heidelberg was one of his first stops, and he studied internal medicine under Dr. Erb. Later he went to Bern.

Dr. Olmsted returned to this continent, and studied at Johns Hopkins and at the Mayo clinic, making trips abroad. Returning again to Hamilton, he practised surgery until about two years ago. For some years medical superintendent of the Hamilton General Hospital, he was closely identified with the growth of that municipal institution from the date of his graduation, and was instrumental in founding a school of nursing there.

Dr. Olmsted was a Fellow of the Royal College of Surgeons of Canada, and a Fellow of the American College of Surgeons. His contributions to medical science were recognized in December of 1934, when he was presented with a life membership in the Hamilton Academy of Medicine.

Surviving are his wife, Edith Olmsted; two sons, Archie Olmsted and Dr. Alexander I. Olmsted, both of Hamilton; a daughter, Mrs. George Hendrie.

Dr. W. S. T. Connell, President of the Hamilton Academy of Medicine pays tribute to Dr. Olmsted's memory in the following words.

''The late Dr. Olmsted was held in the highest esteem by his professional brethren in Hamilton. He possessed qualities in mind and body which fitted him in the highest degree for his profession, while the strict and conscientious attention which he paid to all cases early endeared him to his patients. Possessed of an accurate memory, stored with interesting incidents, both medical and social, he was a delightful companion, and will be greatly missed at medical gatherings in Hamilton.''

Dr. E. A. Arseneau, of Richibucto, N.B., died recently at the age of 42. He had been in poor health for some months. He was born at Balmoral and was a graduate of Laval University, Quebec (1918). He was Medical Officer at the Port of Richibucto and Medical Officer for the Indian reservation in that district.

Dr. Robert James Campbell, of Winnipeg, aged 75, died, almost literally in harness, at his home, on January 29, 1937. On the previous day he had made his rounds and seemed his usual cheerful self. He graduated from the Manitoba Medical College in 1891, and before coming to Winnipeg practised in Rapid City, Carnduff, Sask., and Boissevain, where he was a partner of the late Dr. Lawrence Shaffner, former M.P. and Senator; he had practised in Winnipeg for the past fifteen years. Dr. Campbell was interested for many years in the work of the College of Physicians and Surgeons, was President in 1926-27, and at the time of his death was Chairman of the Discipline Committee of the College. St. John's Cathedral, in which he served as vestryman, was another of his interests. He was a doctor of the old school, with all that that implies of integrity and conscientiousness.

Dr. Reginald Eugene Sproston Challener, of Toronto, died on January 21, 1937. He was born in Toronto in 1900 and was a graduate of the University of Toronto (1924). Dr. Challener was head of the Moss Park district of the Toronto Department of Public Health.

Dr. Alexander Beaton Chalmers, of Fort Erie, Ont., died on January 8, 1937, aged 78 years. He had practised in the United States but had been located in the Niagara district for about fifteen years. He was a graduate of Glasgow University (1881). He is

survived by his widow and one son, J. Stuart Chalmers, of Fort Erie.

Dr. Alexander Forin, of Edmonton, Alta., died on January 2, 1937, at the age of 79 years. He was a graduate of Queen's University (1884). He first came to Edmonton in 1902 and was actively identified with the life of this city since then. He was one of the early presidents of the Alberta Medical Association.

Dr. Archibald Joseph Kilgour, of Kingston, Ont., died on January 28, 1937. He was born in 1893 and a graduate of the University of Toronto (1922), and was a member of the Provincial Department of Health. He is survived by his widow, May Urquhart Kilgour.

Dr. William Alexander Chisholm Macdonald, of Windsor, Ont., died on January 24, 1937, at the age of sixty-one.

Dr. Macdonald was born in Windsor, the eldest son of the late Colin Macdonald, one of the founders of Bartlet, Macdonald & Gow, Limited. After receiving his early education in Windsor public and high schools, he graduated in medicine from the University of Toronto in 1899. He then decided to become an eye, ear, nose and throat specialist and went to Europe for post-graduate work at Edinburgh, Glasgow, Berlin and Vienna. Upon his return to Canada, Dr. Macdonald opened an office in Toronto, but after two years in that city returned to his native Windsor.

During the Great War he earned for himself additional laurels for his service with the Canadian Army Medical Corps. From 1916 to 1919 he was stationed at Le Tréport, France, where he was in charge of the eye department of Canadian General Hospital, No. 2.

Surviving are his widow, Annie E. Macdonald, daughter of the late Mr. and Mrs. Joseph Goodchild, of Collingwood; three sons, Alastair, a medical student at the University of Toronto; Colin, a mining engineer at South Porcupine, Ont., and Ian, a graduate of the University of Toronto and now a student at Osgoode Hall.

Dr. George Alexander McQuibban, of Alma, Ont., Liberal member of the Ontario Legislature for North Wellington, died on February 1, 1937.

Born at Ingersoll, in 1886, the son of James and Helen (Robertson) McQuibban, Dr. McQuibban was educated in Harriston and the University of Toronto, whence he graduated in medicine in 1911. He commenced practice almost immediately in Alma, in partnership with his brother, James. That partnership continued, although since 1931, Dr. James McQuibban had made his headquarters in Elmira.

Dr. McQuibban was unmarried.

Dr. Wilson Montgomery, of Embro, Ont., died on January 7, 1937. He was born in Northern Ireland in 1856, and a graduate of Trinity University (1891). His widow and one brother, William, survive.

Dr. Robert Moore, of Fort Frances, Ont., died on November 7, 1936. He was born in 1864 and was a graduate of Trinity University (1896).

Dr. Walter Henry Pentz, of Halifax, N.S., died on January 10, 1937. He was born in 1889 and a graduate in pharmacy and medicine (1928) of Dalhousie University. He is survived by his widow and two daughters, Irene and Sylvia.

Dr. David A. Stewart, Superintendent of Ninette Sanatorium, Manitoba, and an internationally known authority on tuberculosis, died on February 16, 1937, in hospital in Winnipeg after a long illness. He was 63 years of age.

Born at Fletcher, Kent County, Ont., Dr. Stewart received his early education there and at Chatham, Ont. His parents moved to Morden, Man., and he entered the University of Manitoba, graduating in arts in 1899. He intended to enter the Presbyterian ministry, but his health became impaired and he turned to medicine. He obtained his degree in 1906 and two years later was placed in charge of the projected establishment of a tuberculosis sanatorium in Manitoba.

Dr. Stewart was President of the Manitoba Medical Association in 1926 and the same year was Vice-president of the American National Tuberculosis Society. He was an active member of the Canadian Medical Association and the Chairman of its Committee on Ethics. Before his death he had been engaged on a proposed revised Code of Ethics. As a writer, he was picturesque and incisive, and his contributions were always thoughtful and well received.

Mrs. Stewart, the former Ida Kate Bradshaw, one of Canada's outstanding women advocates of world peace, died last November.

Dr. William Henry Woods, of Mount Brydges, Ont., died on January 27, 1937, in his 66th year. He was a graduate of Trinity University (1898). He is survived by his widow, formerly Isabel Anderson.

News Items

British Empire

The Indian Medical Service.—It has been called to our attention that medical graduates may be interested in the Indian Medical Service, which offers a permanent career with wide opportunities for experience in clinical and preventive medicine, and in research. Candidates must be under 32 years of age and of pure European descent. Pay starts at about $235 per month for overseas pay for the first year; captains receive from $288 to $456 per month; majors from $492 up to $600, and lieut.-colonels from $690 (until twenty-three years of total service is complete) up to $816 per month. Leave is given at reasonable intervals and adequate leave pay provided. Allowances of 12 shillings a day for study in Great Britain or of $7.50 per day for study in the United States or Canada are available for post-graduate study. Pensions range from $1,860 after seventeen years of service to $3,720 after twenty-seven years of service; additional pensions ranging from $325 to $1,750 per annum are admissible to officers who have held administrative appointments.

For those who desire to leave the Service, gratuities of $5,000 after six years of service, or of $7,500 after twelve years of service, are admissible. Free passage from the United Kingdom or Canada, and also for a certain number of leaves, is provided. We understand that the India Office at Whitehall, London, S.W.1, has made arrangements whereby candidates may be interviewed in Canada at stated intervals, thus saving the expense of the journey to England to appear before the Selection Committee at the India Office. This Service would seem worthy of investigation by the recent graduate in medicine. It would appear, however, that the Service during the junior years, because of the various outlying posts to be covered, would be more suited to a single man than one with a young family.

Great Britain

Old Students of Moorfields.—It is probable that during the time of the Coronation some old students of Moorfields from overseas will be in London. A short series of demonstrations that they may like to attend is being staged, and the following program

is being arranged: (1) Exhibition of ophthalmoscopic conditions, Tuesday, May 4th, at 5.30 p.m.; (2) exhibition of slit lamp cases, Thursday, May 6th, at 5.30 p.m.; (3) operation demonstrations: (*a*) cataract, (*b*) glaucoma, (*c*) detachment, (*d*) squint; a selection of the above on the mornings of Monday, May 3rd, Wednesday, May 5th, Friday, May 7th; (4) orthoptics, Tuesday, May 4th, at 10 a.m.; (5) pathological exhibitions, Wednesday, May 5th, at 5.30 p.m.

A welcome is assured to any of our Canadian friends, and it would make arrangements easier if they would be so good as to send us notice of their intention to attend the course:—Charles Goulden, Dean of the Medical School, Royal London Ophthalmic Hospital.

The Senatus Academicus of the University of Edinburgh, on the recommendation of the Faculty of Medicine, has awarded the Cameron Prize for 1937 to J. Bertram Collip, A.M., Ph.D., D.Sc., M.D., F.R.S., Professor of Biochemistry in McGill University, Montreal.

The Cameron Prize has a value of approximately $1,000. The conditions under which it is awarded are set forth in the calendar of the university as follows: "Awarded annually, if the Senate thinks an award justified, to a person who in the five years immediately preceding has made any highly important and valuable addition to practical therapeutics.

"The winner of the prize may be required to deliver a lecture or course of lectures on, or to publish as the Senate may prescribe, an account of his contribution to practical therapeutics."

Alberta

A sectional meeting of the American College of Surgeons is to be held in Edmonton, on March 24, 25, 1937, to which all physicians are invited. No registration fee will be charged and everybody is welcome. Dr. J. O. Baker, of Edmonton, is the Secretary of the Committee and he will gladly furnish further information. While we have not the names of those who will give lectures and clinics we are assured they will be of outstanding ability, and the whole meeting will be such as would warrant a very large attendance of the members of the profession.

At the present time, there are over 530 members of the Alberta profession taking the *Canadian Medical Association Journal*, for which the Alberta Division pays the National Association a lump sum. It was felt by some that under the arrangement with the National body it would be possible to have clinics, hospital staffs, and other groups accept fewer copies so long as there was sufficient copies available at the institution or group offices. Such was found not to be acceptable as most men desired to read their copies in the quiet of their homes. This is a tribute to the quality of the *Journal* which is no doubt appreciated by the Editors.

The burden of caring for persons who are on relief and ill still rests on the medical profession. During the six months' period ending with November last, 100 Calgary physicians made 40,885 calls, performed 1,271 operations, and attended 440 confinements, and for all this work they received $12,000. Had they been paid for the operations alone at the rate of $10.00 each, the confinements and all the visits were done for nothing; had the operations and confinements been done for nothing, it left a rate of 30 cents each for the calls made.

In Edmonton, the group hospitalization plan is working so well that the Committee in charge has decided to use display signs on the bill boards, lauding its merits, so "He who runs may read". If men in industry come in in groups, their rate is 60 cents per month, a lower rate for the wife, and still less per month for the children. It provides 30 days' hospitalization if needed, but so far the average has been less time than ten days.

The Board of Directors of the Alberta Division, Canadian Medical Association met in Edmonton on February 18th. Among the items of business was the question of the "Refresher Course" for the spring at the university, fixing the date of the annual meeting which is to be held some time in the fall at Edmonton, and appointing the necessary committees to look after Convention arrangements.

This is still the medical man's worry. During the depression which is still with us, municipal councils are inclined to increase the services wanted, while decreasing the payment for the same. If the physician owns property that seems to be further reason to reduce his remuneration. Men against their best judgment are being obliged to submit to unreasonable conditions. G. E. LEARMONTH

Manitoba

THE SANATORIUM BOARD OF MANITOBA.—The annual meeting of the Manitoba Sanatorium Board was held in the Central Tuberculosis Clinic on February 2nd. In the absence from illness of Dr. D. A. Stewart, the Superintendent, Dr. E. L. Ross, the Assistant Superintendent, presented the report of the medical activities of the Sanatorium.

The Sanatorium at Ninette, in addition to treating a daily average of 270 patients, dealt with 1,014 people coming in for diagnosis or re-examination, carried the responsibility and management of the Travelling Clinic, and conducted the Christmas Seal Campaign. The total admissions for the year were 325, of which 30 per cent came through the Central Tuberculosis Clinic. Of the total 75 per cent were new cases and 25 per cent re-admissions. Eighty-two per cent had pulmonary tuberculosis, 12 per cent non-pulmonary, and 6 per cent were non-tuberculous. Of 323 patients discharged, 66 per cent were well on the way to a cure, 8 per cent improved, 7 per cent unimproved, and 19 per cent dead. The average length of treatment was 385 days.

The past year was the second in which chest surgery had been done in the institution, and it was felt that the safety and usefulness of surgical methods as treatment were much greater when applied without removal of the patient from the Sanatorium. Seventy-nine major surgical chest operations were performed during 1936. Of those who had had thoracoplasty for more than six months 75 per cent were either sputum- or tubercle bacilli-free.

In the laboratory 10,767 tests were done, an increase of 3,435 over the previous year. Special attention was given to the examination of urine, with a new method of concentration to facilitate the search for tubercle bacilli. Guinea pigs continued to give the most reliable proof of tuberculosis, especially when suspected urine was injected.

The x-ray department at Ninette made 6,788 examinations, 4,418 on Travelling Clinics, and 2,370 at the Sanatorium, at an average cost of 50 cents per examination for film and chemicals.

At the Sanatorium 388,653 meals were served, at an average food cost per meal of 11 cents.

The Travelling Clinics begun in 1926 were continued. Over 250 clinics have been held at 70 different centres. In 1936 39 clinics were held and 4,505 patients examined.

The high incidence and death rate from tuberculosis among the Indians continues to excite the interest of the Board. Since 1930 members of the Sanatorium staff have examined and x-rayed 1,880 Indians, 409 from reserves, 101, or nearly 25 per cent,

were found to have tuberculosis. In Indian Residential Schools less than 5 per cent of the children had the disease.

The Manitoba Minister of Health is asking the Federal Department of Indian Affairs to finance a survey of the whole Indian population in reference to tuberculosis. If this proposal is entertained the survey will be made by the travelling clinics of the Sanatorium Board in cooperation with the Manitoba Department of Health. It is evident that the battle against tuberculosis has become a very elaborate and intricate affair.

Joseph Doupe, of Winnipeg, M.D.(Man.), M.R.C.P. (Lond.), has been awarded the Senior Fellowship offered by the Sir Halley Stewart Trust tenable in the Neurological Research Unit at the National Hospital for Nervous Diseases, Queen Square, London. The Fellowship was made possible by the Sir Halley Stewart Trust placing at the disposal of the Medical Research Council of Great Britain a sum of £500 a year for three years, and Dr. Doupe becomes first holder. It is a fitting award, since Dr. Doupe's grandfather was the late Dr. David Young, pioneer psychiatrist in Manitoba and the first Superintendent of the Hospital for Mental Diseases at Selkirk.

ROSS MITCHELL

New Brunswick

At the January meeting of the Saint John Medical Society, Dr. L. DeV. Chipman was the guest speaker. His subject was "Squint". The speaker spoke of the commoner causes of squint, combinations of nerve paralysis and muscle weakness, most commonly seen, and spent a considerable time on treatment and the accurate diagnosis of the various conditions. At first glance this subject appeared to be one chiefly appreciated by eye specialists. However, Dr. Chipman was able to make the application of his remarks so pertinent that a large audience of general practitioners was closely interested throughout the address.

Dr. C. W. McMillan, Medical Health Officer in the southern part of the province, states that there is no undue seasonable prevalence of influenza at present. There is however a long-continued, mild epidemic of mumps and measles, fairly general throughout the province.

Lieut.-Col. D. C. Malcolm, M.C., C.A.M.C., was the speaker at the Annual Meeting of the Association of Officers of the Medical Services of Canada. The subject of his address was "The recent pilgrimage to Vimy".

Lieut.-Col. R. A. Hughes, E.D., C.A.M.C., has been re-elected President of the Institute of the United Services at Saint John.

Lieut.-Col. V. D. Davidson, A.D.C., C.A.M.A., has been elected president of the Garrison Officers' Mess at Saint John.

Dr. J. A. McLaughlin, recently practising at Perth, has been appointed Assistant Medical Officer of the Workmen's Compensation Board.

Dr. C. O. McKay sails this month to continue his post-graduate studies in orthopædics in Great Britain.

A. STANLEY KIRKLAND

Nova Scotia

A proposal has been put forward to abolish the old quarantine station on Lawlor's Island and to replace this by a new hospital containing six to eight beds on the site of the old Trachoma Building at Rockhead. The city of Halifax will be asked to cede a piece of land surrounding the proposed site. In turn, the city will be provided with facilities for the care of such as suffer from smallpox. The cost of the building will be borne by the Federal authorities, but the city will be allowed to use the hospital.

The Soldier's Memorial Hospital at Middleton, which was opened on Armistice Day, 1921, has been considerably enlarged in recent months, and considerably more accommodation for patients is now available. Dr. J. A. Sponagle, one of the oldest practioners in the province, was largely responsible for the erection of this hospital.

A brief presented before the commission investigating the Workmen's Compensation Act, by Dr. H. K. MacDonald, on behalf of the Committee of the Medical Society of Nova Scotia, pleaded for an increase in fees paid to doctors and for the abolition of the thirty day limit for medical aid as stipulated by the Act.

The report of Dr. G. C. MacDonald, medical officer on the departmental vessel *Arras* that accompanies the fishing fleet to the Grand Banks, shows that 398 fishermen were treated during the season. In only one instance was hospital treatment necessary. Gastric disturbances formed the majority of the ills. Early in the season there were a number of cases of upper respiratory infections. Sometimes he visited Newfoundland outposts where there were no medical practitioners.

Many medical graduates from Dalhousie University will learn with regret of the death of Dr. D. Fraser-Harris, in London, on January 3rd, at the age of sixty-nine. For fifteen years he occupied the chair of physiology at Dalhousie University. Since his retirement from teaching in Canada he has been devoting himself to research work in London.

Dr. Walter H. Penz, who graduated in 1928, died on January 10th, at the age of forty-seven. Before engaging in the study of medicine he had graduated as a pharmacist from the Nova Scotia College of Pharmacy.

Dr. T. R. Johnstone, medical officer for Colchester County, in his annual report, stresses the need for a Tuberculosis Annex to the County Hospital. He also strongly advised taking advantage of the visits of the Provincial Government Engineer to different localities to advise on sanitation questions.

Dr. Ross Miller, director of the medical services for the Department of Pensions and National Health, Ottawa, visited the province during the past month on departmental business.

Dr. T. A. Lebbetter, of Yarmouth, was elected President of the Dalhousie University Alumni Association organized there in connection with the reunion plan for August, 1938. This was the first of several distinct Dalhousie groups which will be organized throughout the province.

The Honourable Angus L. Macdonald, Premier of Nova Scotia, sent a telegram to Dr. Thomas E. Parran, Surgeon-general of the United States Public Health Service, offering the aid of the medical and nursing unit to help in the flood-stricken areas.

N. B. DREYER

Ontario

Florence M. LaRush, B.A. (Queen's), M.A. (Toronto), died on January 24th, following an operation. Miss LaRush graduated from the Department of Botany, University of Toronto. She was associated for thirteen years with the Department of Soldiers' Re-establishment at Christie Street Hospital and with the Connaught Laboratories.

Working in the field of allergic preparations, Miss LaRush was successful in collecting and preparing for the medical profession pollens and other substances of Canadian origin. Her work in this field since 1913 has been of great assistance to physicians.

Dr. P. A. T. Sneath, D.P.H., Captain R.C.A.M.C., who was for some years attached to the Colonial Medical Service in West Africa, now a lecturer in Hygiene and Preventive Medicine in the University of Toronto, and Research Associate in the Connaught Laboratories, has been selected for the post of first assistant in the Government Medical Health Service of British Guiana, leaving on March 1st.

The Willit Hospital at Paris, Ont., has received a grant of $15,000 from Brant County Council to provide much needed extension and equipment.

More than $25,000 has already been donated to St. Mary's Hospital, Timmins, towards the building of the new nurses' home, on which some $50,000 will be expended.

J. H. ELLIOTT

Saskatchewan

The Saskatoon and District Medical Society held their annual meeting after a dinner at the City Hospital. Dr. S. R. Laycock, Professor of Educational Psychology, University of Saskatchewan, spoke on "Diagnostic procedures in behaviour problems". The following officers were elected: *President*, Dr. L. H. McConnell; *Vice-president*, Dr. J. J. Finn, Dundurn; *Secretary*, Dr. Medley Hazen; *Executive*, Drs. Lloyd Anderson, G. H. Hames, Saskatoon Sanitorium, and J. A. Valens.

At a recent meeting of the Regina and District Medical Society it was reported that in 1936 the relief recipients of Regina had received medical services from the physicians to the value of $75,000, for which the physicians have been paid $24,000 by the city.

LILLIAN A. CHASE

General

Synopsis of the Report of the Banting Research Foundation for the Year 1935-1936.—In all some 22 different grants were distributed to workers in the laboratories of the universities throughout Canada during the period covered by this report. A number of them were for comparatively small sums. For the first time in the history of the Foundation there were a larger number of grants made to workers in the University of Toronto than to other universities, and yet more grants were refused applicants in Toronto than in other universities. This is probably to be explained by the fact that the financial depression has perhaps affected the University of Toronto less than some of the other universities, though it has had the effect of increasing the number of well qualified men who are unable to find posts, and who consequently make application to the Foundation to give them the means by which they can carry on their investigations. It is to be hoped that the raising of the depression will be followed by a more equitable distribution of the grants.

As a result of a grant made to Dr. A. C. Abbott and Dr. James Prendergast, University of Manitoba, the careful study of the effect of pregnancy on the state of the thyroid gland was published. The grant to A. J. Cipriani, McGill University, was continued in order to enable him to complete his study of the methods of electrical recording of heart and respiration rates. The summer grant made to B. K. Coady and D. L. MacIntosh, Dalhousie University, following small grants made in previous years, resulted in the publication with Prof. Mainland of a series of four papers dealing with the character and counts of white blood cells with proper statistical analysis of their variation. The grant to K. A. Evelyn, McGill University, was continued for another year in order to make clinical application of his method of photo-electrocolorimetry. The description of this apparatus has now been published and already various laboratories have adapted it for use, and there is no doubt that a valuable tool has been put in the hands of biochemical workers. Papers on the clinical application of the instrument are already in press. A grant to Dr. L. Farber, Toronto, enabled him to make the preliminary steps in a study of the metabolism of certain of the infective bacteria. A grant to Dr. A. W. Ham, Toronto, enabled him to complete his study of the histological changes produced by chronic vitamin C deficient diet. This paper is ready for the press. A grant to C. O. Hebb, McGill University, resulted in a paper showing the relationship between external pancreatic secretion and the glucose content of the blood stream. A grant made to A. H. R. Smith, Toronto, led to a minor paper dealing with the anæsthetic effects of some substituted furans and the development of a method of electrical recording of pulse pressure. Grants made to Prof. L. Irving and K. M. Robertson, Toronto, enabled further progress to be made with the study of the effect of the carbon dioxide anhydrase, particularly in regard to the production of bone. The grant made to Dr. S. H. Jackson, Toronto, enabled a study to be made of the effect of skin infections on the glucose tolerance of animals fed both a high and a low carbohydrate diet. This material is also ready for press. A grant to Dr. E. M. Macdonald, Toronto, for the study of the transmission of immunity to the infant, is making progress. A grant to C. B. Stewart, Dalhousie, for a study of the accuracy of percussion has enabled him to gather a sufficient number of cases for a statistical study. A grant made to Dr. T. S. Perrett, Toronto, enabled further progress to be made on the effect of heparin on the prevention of thrombosis. A grant made to B. Schachter, Toronto, working under Prof. G. F. Marrian, enabled them to publish a short paper on the œstrogenic substances in mares' urine, which shows that there is less variation in free and combined substances during pregnancy in this species than occurs in man. A grant made to Dr. M. C. Watson, Toronto, has led to the publication of the effects of sex hormones (supplied in part by Prof. Marrian) on the disturbances of the menstrual cycle. A grant made to Dr. F. Smith, McGill University, has led to the publication of a paper pointing out that strains of pneumococci exist which can grow under anaerobic conditions. A grant made to Dr. S. Weinstein, Toronto, led to the publication of a paper on the purification and assay of one of the sex hormones obtained from human urine during pregnancy. The grant to Dr. J. K. W. Ferguson, University of Western Ontario, for apparatus enabled him to complete a study of the carbamic compounds which are so important in the transport of carbon dioxide in the blood stream; a study of very considerable physiological importance. A small grant was made to Dr. M. J. Wilson, Toronto:—V. E. Henderson, D. T. Fraser, Honorary Secretaries.

Dr. C. C. Macklin has received word that he has been elected unanimously to membership in the Anatomische Gesellschaft, which is one of the most important anatomical societies in the world. Two other British subjects were elected to this society at the same time, Dr. John Beattie, Curator of the Museum

of the Royal College of Surgeons of London, and Dr. Thos. B. Johnston, Editor-in-Chief of the Gray's Anatomy and Professor of Anatomy in Guy's Hospital. Dr. Macklin has been a member of the Anatomical Society of Great Britain and Ireland for twelve years and of the American Association of Anatomists for twenty-two years. He served on the Executive Committee of the latter organization for four years and last spring presided at a Round Table Conference on Alveolar Epithelium under its auspices. He has been a Fellow of the Royal Society of Canada for twelve years. In September last he gave a paper and demonstration at the fourth International Congress of Anatomists at Milan, Italy.

Dr. Madge Thurlow Macklin, of the University of Western Ontario Medical School, was one of the twelve speakers invited from America and Europe to give addresses at the three-day Cancer Symposium held under the sponsorship of the University of Wisconsin on September last. Her first address was entitled "The rôle of inheritance in cancer in man". At the Round Table Conference, which was part of the Symposium, she gave an address on "The relative values of chronic irritation and heredity in producing cancer". This Conference was shared in by Dr. C. C. Little, Director of the American Society for the Control of Cancer, and by Prof. Leiv Kreyberg, of the University of Oslo, Norway. Dr. Macklin also gave two addresses in Philadelphia on December 2, 1936; one on "The brighter side of the cancer problem", when she was luncheon speaker at the Cancer Forum sponsored by several organizations, among them the Lankenau Hospital Research Institute and the American Society for the Prevention of Cancer; the second was given before the Philadelphia County Medical Society on "Heredity as a major factor in causing cancer". Prof. J. McFarland, McManes Professor of Pathology at the University of Pennsylvania, also spoke on cancer on that occasion.

The Vienna Association of Physicians announces a special post-graduate course to commemorate the hundredth anniversary of its foundation, which will take place from May 19th to 29th. An excellent program has been prepared. The fee is 25 schillings. All information can be obtained from the Kursbüro der Wiener Medizinischen Fakultät, Allgemeines Krankenhaus, 1, Hof., Wien IX, Austria.

Fellowship in Tuberculosis. — The Tuberculosis Division of the Provincial Board of Health, British Columbia, again offers a Fellowship in Tuberculosis for the year 1937-38, this to commence on July 1, 1937. This Fellowship is awarded for the Vancouver Unit, and is for $1,200. Applicants must be physicians qualified to practice in British Columbia, and full time must be devoted to the work.

Facilities available.—The Vancouver Unit is attached to the Central Headquarters of the Tuberculosis Division of the Provincial Board of Health, so that all records relative to tuberculosis cases in the province are available through this Centre. All records from each Unit, be it an institution, travelling or stationary clinic, social service or district nursing department, are on the punch-card system, and are therefore readily available for study.

The Unit is a closed department with its own medical staff. There are 255 in-patient beds for active treatment type of cases, and a large out-patient clinic, examining approximately 1,000 patients a month. Complete medical service is given, including chest surgery, genito-urinary service, eye, ear, nose and throat service, bronchoscopy, laboratory services, etc.

Applications will be accepted until March 31, 1937. W. H. Hatfield, M.D., Provincial Medical Director, Tuberculosis Control.

Book Reviews

The Physiological Basis of Medical Practice. C. H. Best, M.A., M.D., D.Sc., etc., Professor of Physiology and Head of the Department of Physiology, and N. B. Taylor, M.D., F.R.C.S.(Edin.), F.R.C.P.(C.), etc., Professor of Physiology, University of Toronto. 1684 pages, illustrated freely. Price $10.00. William Wood & Co., Baltimore, 1937.

A book of this type has long been overdue, and this particular work is indeed welcome. Medical educationists have for years insisted on the importance of the so-called basal sciences—chemistry, physics, and biology — and the pre-clinical subjects, anatomy and physiology, in the training of the medical student. They are also aware of the desirability of linking up these with the clinical work of the later years. But, is this being done? Not so well as it might be, we fear. The value of these preliminary subjects in begetting a knowledge of research methods and in fostering the practice of logical analysis and deduction will be admitted by all. This is well, so far as it goes, but it is not enough. Unless the applications to clinical medicine of the knowledge acquired in these earlier years are indicated the student may well wonder what it is all about. In regard to the liaison between the preliminary and the clinical studies the teacher may also be at a loss. Both student and teacher may profit, then, by the help which such a book as Best and Taylor have provided. The authors have "endeavoured to write a book which will serve to link the laboratory and the clinic, and which will therefore promote continuity of physiological teaching throughout the pre-clinical and clinical years of the undergraduate course". In this purpose they have succeeded admirably.

The work is most comprehensive—there are 8 sections and 73 chapters; there is a good index, and an adequate bibliography at the end. The various sections are introduced with a discussion of the applicable facts in respect of the anatomy and physiology of the various systems, and the related clinical states are considered as examples of disordered function. A useful feature is that in the table of contents the clinical conditions referred to are indicated by italics, thus making it easy to find any particular topic. As a sample of the method adopted we may cite one of the shorter sections, that on Respiration. Chapters are to be found on The Mechanics of Respiration; The Air of the Lungs; The Physical Principles governing the Respiratory Interchanges; The Transport and Delivery of Oxygen to the Tissues; The Carriage of Carbon Dioxide by the Blood; The Control of Respiration; and Anoxia. The topics of clinical interest incidental to these discussions are Artificial Respiration; Pneumothorax; Collapse of the Lung; Bronchiectasis; Dyspnœa of Various Types; Anoxia; Mountain Sickness; Caisson Disease; Asthma; Emphysema; Atelectasis; Shunt; Carbon Monoxide and Cyanide Poisoning; Cyanosis; Oxygen Therapy. And so with all the other sections. Everywhere the relation between normal and abnormal function is brought out, with a wealth of illustration and appropriate references to the literature.

The book will be of great value to the medical student, to be taken in conjunction with his formal didactic lectures and laboratory experiments, and, later, when he is being introduced to clinical problems; it will also interest the clinical teacher who wishes to brush up his knowledge of physiology and its applications. For these purposes the work cannot be too highly commended. The high standing of its authors is in itself a guarantee of the excellence of its contents. We may add that the practitioner who is some years out of college will also find this book instructive and stimulating.

Neurological Surgery. Loyal Davis, M.S., M.D., Ph.D., D.Sc., Professor of Surgery, Northwestern University Medical School. 429 pages, illustrated. Price $6.00. Lea & Febiger, Philadelphia, 1936.

This very excellent volume is primarily presented to acquaint the general practitioner with the advances which have been so rapidly made in the past few years in this highly specialized field of surgery. The author has presented the subject clearly and simply, with a view of removing the general idea that neurosurgery is too complicated to be comprehensible to the average general man. Among the many interesting features in this work is his comparison of his own statistics in regard to the brain tumour group with those of Harvey Cushing. His chapters upon craniocerebral and spinal injuries are extremely sound and in view of the increasing frequency of these injuries should be read by every man practising medicine.

The book is very well compiled and profusely illustrated. The reviewer knows of no volume which so thoroughly and simply presents this subject.

Textbook of Surgery. John Homans, M.D., Clinical Professor of Surgery, Harvard Medical School. Fourth edition, 1267 pages, illustrated. Price $8.00. C. C. Thomas, Springfield and Baltimore, 1936.

Editions of this work follow each other rather quickly, but the desire is evident to keep the book well up to date. One of the main features in this edition is a new chapter on amputations and plastics. This chapter will be supplied separately to those who wish to add it to their previous editions. Various minor additions and changes have also been made.

Diseases of the Eye. Sir John Herbert Parsons, C.B.E., F.R.C.S., F.R.S. Eighth edition, 705 pages, illustrated. Price 18s. J. & A. Churchill, London, 1936.

This textbook has been marked by such excellence through its many previous editions that it needs very little introduction. It is well and concisely written; the many illustrations are well chosen and well executed. The rapid advances during the last few years in our knowledge of sympathomimetic and parasympathomimetic drugs have rendered necessary this new edition within the short space of two years. Likewise, the author's comments on the treatment of detachment of the retina are timely. There is no one better qualified than Sir John Parsons to evaluate these matters. This he does impartially and yet critically. Indeed, the volume contains much of value not only to the student but to the mature specialist.

Principles of Biochemistry. Albert P. Mathews, Andrew Carnegie Professor of Biochemistry, University of Cincinnati. 512 pages. Price $4.50. William Wood & Co., Baltimore, 1936.

The new "Mathews" is, as stated on its dust-cover "not a revision or condensation of the author's long-famous standard Textbook of Physiological Chemistry, but a completely new work". Although this volume has many admirable features, not the least of which is its remarkable "up-to-dateness", it is difficult to recommend it unreservedly as a textbook for medical students. The average student requires a text for use as a reference book with which to correct and supplement his lecture notes. Professor Matthews' easy and conversational, but somewhat verbose, style has produced a book which is delightful and unusually stimulating to read, but which is unsuitable for reference purposes. It is difficult to escape the conclusion that the average medical student, who of necessity demands of a book that it should contain the maximum of knowledge arranged systematically in the minimum of space, would be irritated by this text. On the other hand one has no hesitation in wholeheartedly recommending it to the more leisured student who regards biochemistry as something more than a mere examination subject. The author has quite obviously set out to capture his reader's interest and in this he succeds admirably. Perhaps he is somewhat too prone to be dogmatic where the state of our knowledge does not justify dogmatism, but after reading his book one is left with the conclusion that as a lecturer Professor Matthews can have few equals.

The arrangement of the subject matter is unusual and there is much unnecessary and space-consuming repetition, but with a few notable exceptions the whole field of biochemistry is covered in an unusually adequate manner for a book primarily intended for medical students. It is therefore surprising to find that the subjects of hydrogen-ion concentrating, carbon dioxide transport in the blood, and energy metabolism do not receive the space or attention which their vital importance merits. The writing is in most places remarkable for its clarity, but in one instance, at least, the author, in his endeavour to explain his ideas in a simple manner, has only succeeded in achieving obscurity, as the following will show.—"The pH is the exponent of 10 in the denominator when the concentration of hydrogen ions is expressed as a normal solution divided by 10 raised to some power. The pH is the power to which the 10 is raised".

The Clinical Use of Digitalis. Drew Luten, A.B., M.D., Associate Professor of Clinical Medicine, Washington University School of Medicine. 226 pages. Price $3.50. C. C. Thomas, Springfield and Baltimore, 1936.

The last decade has seen some improvements in the treatment of heart disease, the most noteworthy perhaps, being a more accurate dosage with digitalis. Dr. Luten has thought it worth while to reconsider the whole problem and this volume of 226 pages is the result. He pays his tribute to the father of digitalis, William Withering, and shows how thoroughly he studied the drug. For example, Withering recognized that the vomiting following its administration "does not take place for many hours", and therefore was not due to irritation of the stomach, but was a true toxic effect.

The author considers in a very impartial way the controversy between those who consider that digitalis acts upon the nervous mechanism of the heart in auricular fibrillation, and those who think the beneficial effects are due entirely to the action of the drug upon the cardiac muscle. The weight of evidence is almost entirely in favour of the latter view. Auricular fibrillation is not the reason for giving the drug, but rather the cardiac failure, of which the fibrillation is only a symptom. In fact, in toxic cases such as thyroid disease and rheumatism where fibrillation is present without heart failure, digitalis is not only useless but it may be harmful. Many of the psychoses which are so common in cardiac disease are the result of digitalis, a point worth remembering in the treatment of these cases. He sums up the use of the drug in pneumonia by saying that physicians should withhold digitalis unless some unequivocal indication should present itself.

The book is very readable, because of Dr. Luten's pleasing style. Almost every page has something worth while, for the author's conclusions are based on the evidence he has been able to produce from other writers' and from his own experience. He concludes with a quotation from Wenckebach—"Digitalis treatment is one of the most important and serious duties of the general physician; it demands a great deal of skill, power of observation, keen interest, and experience. A long life is too short to learn enough about this wonderful drug."

Chronic Indigestion. C. J. Tidmarsh, M.A., M.D., F.R.C.P.(C.)., Associate in Medicine, Royal Victoria Hospital, Montreal. 143 pages, illustrated. Price $1.50. Longmans, Green & Co., Toronto, New York and London, 1936.

It is difficult to present medical knowledge to the layman, even the intelligent layman. Dr. Tidmarsh has tried, and has achieved some success, but the average layman is not likely to find his book easy reading. However, if, as is insisted on, he will read the book in cooperation with his medical adviser he will find all that he needs to know about his indigestion. The illustrations are likely to be the least comprehensible part. More line drawings like the frontispiece would probably have been more impressive.

Medicine and Mankind. Lectures to the Laity delivered at the New York Academy of Medicine. Edited by I. Galdston, M.D. 217 pages. Price $2.50. D. Appleton-Century, New York and London; Ryerson Press, Toronto, 1936.

An interesting set of lectures for the laity on medical subjects, by members of the New York Academy of Medicine. Medical men will also find a good deal of useful historical detail amongst these.

Diseases of the Nails. V. Pardo-Castello, M.D. 176 pages, illustrated. Price $3.50. C. C. Thomas, Springfield and Baltimore, 1936.

This brochure, by the former assistant professor of dermatology and syphilology of the University of Havana, is a compact but very complete description of the abnormalities to be observed in the nails and associated structures. These are classified according as to whether they are affections peculiar to these organs, dystrophies, or ungual manifestations of dermatoses, systemic diseases, or congenital affections. A clear account of the anatomy and pathology is given, so that the manner in which nail disorders are produced may be comprehended.

The work is illustrated by numerous and well-selected photographs. In such conditions as are amenable to treatment there is no ambiguity or lack of detail in describing the methods used. The categorical statement that spores are not found in properly prepared fresh nail specimens invaded by mycotic disease should be noted by laboratory technicians who not infrequently report finding spores, which are usually artefacts, fat-droplets, etc. Evidently onycholysis is not so frequently observed in Cuba as a psoriatic complication as it is in this country, where it almost divides the field solely with onychia punctata.

At the end of the book a table of occupations in which diseases of the nails are common, showing those usually occurring in each occupation and giving the manner of their causation, is a valuable addition. Similarly useful is a table of ungual symptoms due to poisons, grouped under the poison named. The bibliography contains almost 200 references, and the index is complete and well-arranged.

While this book will be of primary interest to the dermato-syphilologist, it can be read with profit and should be referred to frequently by the general practitioner and internist.

Disinfection and Sterilization. Ernest C. McCulloch, M.A., D.V.M., Ph.D., Biological Research, Pennsylvania Salt Manufacturing Co. 512 pages, illustrated. Price $5.50. Lea and Febiger, Philadelphia, 1936.

This is a very comprehensive and up to date book on the subject of disinfection and sterilization. Its preparation must have entailed a tremendous amount of work on the part of the author. The book is worth while, were it only for the long list of references to the available literature on the subject, although, by the same token, one is sometimes disappointed in finding that the names of authors mentioned in the body of an article are omitted in the reference list at the end of the chapter.

There are in all 19 chapters, dealing among other things with the history of disinfectants, natural, chemical and physical disinfectants, how disinfectants can be compared, and the dynamics of disinfection. There are also chapters on the acids, the alkalies, the heavy metals, the dyes, phenols, cresols and alcohols. The reviewer is impressed with the value of the special chapters on pasteurization, water purification, and the selection of a disinfectant. The discussion on the destruction of pathogenic spore-bearing organisms is well presented, and many teachers of public health will agree with the author when he "seriously questions the advisability of the home-canning of meats and non-acid vegetables unless a steam cooker is used". Some very interesting tables are included, showing the comparative power of mercurochrome and iodine, which go to prove the superiority of iodine as a skin disinfectant. The index is somewhat surprising—one looks through it in vain for the giant names of Rosenau and Park!

The book can be recommended as a reference book for bacteriologists, and for teachers and students of bacteriology and public health.

Remington's Practice of Pharmacy. Edited by E. Fullerton Cook, P.D., Ph.M. and Charles H. LaWall, Ph.M., Sc.D., F.R.S.A. Eighth edition, 2162 pages, illustrated. Price $10.00. J. B. Lippincott, Philadelphia and London, 1936.

The changes in the practice of pharmacy and in pharmaceutical education in recent years have been met by a thorough revision of this "practice" which in its various editions has served pharmacists and physicians for over fifty years. It is, of course, based upon the United States Pharmacopœia XI and the National Formulary, and contains all formulas official in them. This is only one part of fourteen dealing with pharmacy for the compounder, manufacturer, physician, hospital. It has maintained the high standards set in previous editions.

Memorandum Book of a Tenth-Century Oculist for the Use of Modern Ophthalmologists. First edition in English by Casey A. Wood. 232 pages, illustrated. Published by Northwestern University, Chicago, 1936.

In 1929 the learned translator of this work published an English version of the *De Oculis* etc. of Benevenutus Grassus, the first *printed* work on ophthalmology (Ferrara, 1474). Now he has supplemented his effort by undertaking a similar task for what is the most original and complete of the early *written* treatises on the subject—the *Tadhkirat* of Ali ibn Isa. Dr. Wood, after modestly disclaiming any special facility in the Arabic language, states that he has had the valuable assistance of Dr. Max Meyerhof, of Cairo, and he has also freely made use of the earlier work of such scholars as Hirschberg, Hille, and Pansier. The result is a book which may be accepted as a worthy presentation of Ali ibn Isa, and its value is further enhanced by the addition of numerous comments on the text and an illuminative account of the Arabian ophthalmologists who flourished from 800 to 1300 A.D. Dr. Wood also gives a lengthy list of drugs, plants, and other remedial agents used by the early and mediæval ophthalmologists which is embellished by the addition of a number of illustrations of plants taken from Matthioli's beautiful "Commentary on Dioscorides" (Venice, 1554). The instruments used by the Arabians in the treatment of the eye are also figured and described. Ali's work is based, confessedly, on the Greeks and on the Arabians who preceded

him. His anatomy and pathology is that of Galen, but much of his therapeusis is his own. Many of the principles on which he worked strike us, even at this late date, as highly sensible, and the modern ophthalmologist may gain useful hints from Ali. Indeed, the *Tadhkirat* may be accepted as the best treatise on ophthalmology that appeared up to the time of Kepler, at the beginning of the eighteenth century.

Dr. Wood's translation is beautifully produced and will be treasured by all ophthalmologists and those who are interested in the history of the medical art. But, Oh! Oh! surely the vulgarism "sparrow grass" as a synonym for "asparagus" (despite some slight authority) must have slipped into the text (page 49) "unbeknownst" to the proof reader!

A Textbook of Physiology. H. E. Roaf, M.D., D.Sc., M.R.C.S., L.R.C.P., Professor of Physiology, University of Liverpool. Second edition, 679 pages, illustrated. Price $6.25. Ed. Arnold, London; Macmillan, Toronto, 1936.

This book covers the field of physiology in a manner intended to supply the needs of the medical student. The treatment embraces elementary physiology, some biochemistry, and even a little psychology. The book is short for one of such scope and is necessarily superficial. Description of experimental work is brought into the main stream of exposition in a manner which lends additional meaning to much of the subject matter. This method has the advantage of stimulating interest by translating physiological concepts into isolated demonstrations which the student can grasp. Its danger is that the result may be simply a laboratory manual on the history of physiology. The text in question has not entirely escaped this pitfall. Indeed, much of the experimental work cited is interesting as history but not as physiology. This tendency is evident particularly in the sections on muscle and nerve. Here the author has further emphasized the historical aspects of his subject by citing as references textbooks and monographs of the last century, often to the exclusion of modern works. Such over-emphasis of old and at least partly outdated information may prove a source of confusion to students entering the field of physiology. In several chapters the author leaves the beaten path of textbook exposition and brings a pleasing freshness to his subject. The sections on skeletal mechanics and on physical and mental growth show a welcome originality of treatment. The illustrations are good, many of those showing kymograph records being exceptionally fine. The style is well suited to an elementary text of a synoptic nature.

Lectures on Diseases of Children. Robert Hutchison, M.D., LL.D., F.R.C.P., Consulting Physician to London Hospital. Seventh edition, 452 pages, illustrated. Price $6.25. Ed. Arnold, London; Macmillan, Toronto, 1936.

This book is probably as familiar to the general practitioner as to the specialist. The ease of its style, the comprehensiveness of its teaching, the practical nature of its contents, all commend themselves to those with a medical practice. This last edition brings it well up to date.

Bailey's Textbook of Histology. Edited by Philip E. Smith, Ph.D., Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Ninth edition, 73 pages, illustrated. Price $6.00. William Wood, Baltimore, 1936.

This revision shows several new features, notably the preparation of several chapters by a group of teachers in the Anatomy Department of the College of Physicians and Surgeons, each of whom has taken subjects in which he is particularly interested. The book retains its original plan of being for the guidance of students. The illustrations have been added to and improved. The book retains its high place amongst textbooks.

Starling's Principles of Human Physiology. Edited by C. Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D., Jodrell Professor of Physiology, University College, London. Seventh ed., 1096 pages, illustrated. Price $8.75. Lea & Febiger, Philadelphia, 1936.

A seventh edition of such a well known textbook as this hardly calls for detailed comment. In the three years that have elapsed since the last edition physiology has been added to, largely along biochemical lines. This has involved the re-writing of chapters dealing with such subjects as the vitamines, the hormones, especially the sex hormones, the transmission of nerve impulses, etc. The book remains a first class modern textbook of physiology.

Legal Problems in Medical Practice. D. Harcourt Kitchen, of Gray's Inn, Barrister-at-Law. 232 pages. Price $3.00. Ed. Arnold, London; Macmillan, Toronto, 1936.

This is the first medico-legal work written primarily for the medical man rather than the lawyer that has come to us for some time. As the author points out in his preface, his book is chiefly made up of articles that have appeared before in various medical journals, in particular, the *British Medical Journal*. Accordingly, the book is likely to be of greater value to the medical practitioner in Canada than the average work written by a lawyer for lawyers to meet English needs. Like the latter, it gives a certain amount of space to topics such as the responsibility of midwives and the sale of practices that, because of different conditions, are not of so much interest in Canada, and a few of the conclusions, being based upon peculiarly English statutes, must be applied with care. But other chapters, such as that on The Medical Witness, are as applicable to Canada as to England, and they furnish the best treatment of their subjects that we have yet seen. The introductory chapters on the duties of the physician and surgeon are also deserving of attention, and the later chapter on the ownership of x-ray films gives an excellent discussion of a still disputed subject. The whole book is noteworthy for its peculiarly lucid phraseology and its charm of style.

Poverty and Public Health. G. C. M. M'Gonigle, M.D., D.Hy., B.S., D.P.H., M.O.H. for Stockton-on-Tees, and J. Kirby, M.R.S.I. 275 pages. Price 6s. Victor Gollancz, London, 1936.

The authors in approaching the study of health and disease seek for an understanding through non-environmental conditions. Following upon a presentation of the physical condition of the population based upon the reports on the Ministry of National Service, 1917-1919, and the medical inspection of elementary school children, and certain special investigations, all of which reveal a high percentage of physical disabilities, the authors then proceed to an examination of the records of certain child-welfare centres to supplement the previous findings. The second part of the book is devoted to the question of family budgets. It is shown that " . . . . the overhead costs of living, apart from food, are largely beyond the power of the family to control". The "overhead" includes rent, fuel and light, deductions from wages for social insurance, trade union subscriptions, travelling expenses, household utensils, clothing, etc. In the 141 families investigated 55 per cent of the income is spent on "overhead", leaving 45 per cent available for food, but in actual daily life, the balance available for food is also used for tobacco, newspapers, amusements, emergencies, and so on. The inevitable conclusion is

that of the group studied the majority are unable to purchase an adequate diet.

The book is recommended to those interested in the relationship of social conditions to the public health.

Alcoholism in General Practice. A. E. Carver, M.A., M.D., D.P.M., Medical Director, Caldecote Hall, Thomas Hunt, B.A., D.M., F.R.C.P., Physician to Out-patients, St. Mary's Hospital, London, W. 2, and Sir William Wilcox, K.C.I.E., C.B., C.M.G., M.D., F.R.C.P. 131 pages. Price $2.25. Constable & Co., London; Macmillan Co., Toronto, 1936.

This very handy little book is worthy of commendation. The division of the material into a general section on psychology and management, an excellent discussion of the physical effects of alcoholic excess, and a final brief but adequate presentation of the medico-legal aspects are both attractive and convenient. It is encouraging to note the omission of the customary boring moralistic platitudes and the frank acceptance of the psychogenic factors involved. Outlines of treatment are clearly and succinctly stated, and though one could wish for some more statistical support to bolster up the rather dogmatic statements made the main scheme is obviously sound.

Making no profession to contain anything exhaustive or startlingly new, this excellent handbook may be recommended to the general practitioner, although that person is rarely competent to carry out the psychotherapy which is the essence of any therapeutic attack on the great medical and social problem of alcoholism.

Disability Evaluation. Earl D. McBride, B.S., M.D., F.A.C.S., Assistant Professor in Orthopædic Surgery, University of Oklahoma. 623 pages, illustrated. Price $9.00. J. B. Lippincott, Philadelphia, London and Montreal, 1936.

This volume will fill a large void in the field of surgery, and it is almost indispensable to those engaged in industrial and casualty practices.

In the preface, the author states the purpose of the volume "is to interpret the physiological and mechanical operations arising out of injury to the motor structures of the human body and to reasonably appraise and to evaluate the extent of functional loss as it relates to economic incapacity of the injury". The author adheres closely to his thesis. While many will not agree with his method of evaluation of percentage of disability, yet it seems a workable scheme and will serve as a working basis for further study.

Chapter one is devoted to different compensation laws, and chapters two and three deal with standardizing disability evaluation methods and the examination of a disabled person. Both these last chapters are very important and are carefully done. Chapters four to fourteen deal with a very important subject, and that is "Ankylosis, partial and complete" of the different joints. Chapters fourteen to eighteen deal with fractures of the different bones. In estimating disability in this type of injury, he takes into consideration three factors; first, mechanical and physiological alterations; secondly, functional depreciation; thirdly, physiological compensation and possibility of repair. Each of these points is thoroughly discussed and applied in estimating disability in all types of fractures.

There is a very good chapter on the "industrial back" which also includes a consideration of malingering in accidental cases. Head and nerve injuries are also discussed. The last chapters deal with burns and hernia. Both these chapters are well worth studying.

The book is one that could well be studied before giving an estimate of disability in any particular accident. The author's judgments are temperate and the book deserves a wide circulation.

Manual of Pharmacology. By the late W. E. Dixon. Eighth edition revised by W. A. M. Smart. 483 pages. Price $5.50. Ed. Arnold, London; Macmillan, Toronto, 1936.

This will known guide to pharmacology has been well revised and brought up to date by Dr. Smart. Organic chemistry receives greater attention, as well as clinical effects. Numbers of prescriptions have been added, a decidedly useful innovation.

Food and the Principles of Dietetics. Robert Hutchison, M.D., LL.D., F.R.C.P. and V. H. Mottram, M.A. Eighth edition, 634 pages. Price $6.25. Ed. Arnold, London; Macmillan Co., Toronto, 1936.

An eighth edition of a standard work. The increasing interest in diet has been realized and the book retains its prominent place on the subject of nutrition.

BOOKS RECEIVED

The Treatment of Asthma. F. T. Harrington, M.R.C.S., L.R.C.P. 112 pages. Price 6s. net. H. K. Lewis, London, 1936.

Index of Differential Diagnosis of Main Symptoms. Fifth edition edited by H. French, C.V.O., C.B.E., M.A., M.D., F.R.C.P., Consulting Physician to Guy's Hospital. 1145 pages, illustrated. Price $18.00. John Wright & Sons, Bristol; Macmillan Co., Toronto, 1936.

Body Water. John P. Peters, M.D., Professor of Internal Medicine, Yale University School of Medicine. 405 pages. Price $4.00. C. C. Thomas, Springfield and Baltimore, 1935.

Lobar Pneumonia and Serum Therapy. F. T. Lord, M.D., Clinical Professor of Medicine, Emeritus, Harvard Medical School, and R. Heffron, M.D., Field Director, Pneumonia Study and Service, Massachusetts Department of Public Health. 91 pages. Price $1.00. Commonwealth Fund, New York, 1936.

Change of Life in Men and Women. Marie C. Stopes, D.Sc., Ph.D. 282 pages. Price 6s. Putnam, London, 1936.

The True Physician. W. M. Johnson, M.D. 157 pages. Price $1.75. Macmillan Co., New York and Toronto, 1936.

Therapeutic Uses of Infra-red Rays. W. A. Troup, M.D., Ch.B. Third edition, 148 pages, illustrated. Price 10s. 6d. Actinic Press, London, 1936.

Theory and Practice of Psychiatry. William S. Sadler, Chief Psychiatrist and Director, Chicago Institute of Research and Diagnosis. 1231 pages. Price $12.00. C. V. Mosby, St. Louis; McAinsh, Toronto, 1936.

Live Long and Be Happy. Lewellys F. Barker, M.D. 224 pages. Price $2.00. D. Appleton-Century Co., New York, 1936.

Fundamentals of Human Physiology. Late J. J. R. Macleod, M.D., D.Sc., F.R.S. and R. J. Seymour, M.S., M.D., Professor of Physiology, Ohio State University, Columbus, Ohio. Fourth edition, 424 pages, illustrated. Price $2.75. C. V. Mosby, St. Louis; McAinsh, Toronto, 1936.

Hydrotherapy. Ruth M. Le Quesne, Chartered Masseuse, Bio-Physical Assistant, etc., for C.S.M.M.G. and Mary Granville, State Registered Nurse for C.S.M.M.G. 142 pages. Price $2.00. Cassell & Co., London; McAinsh, Toronto, 1936.